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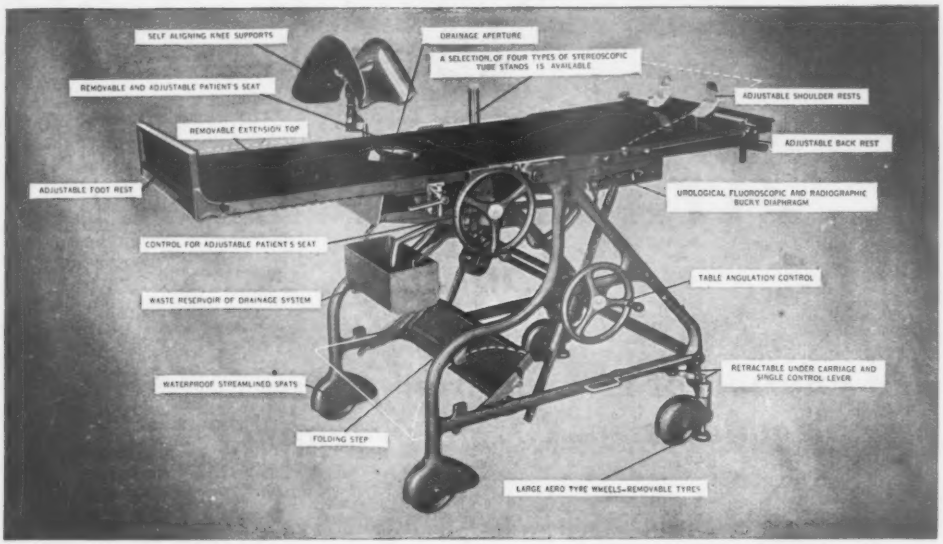
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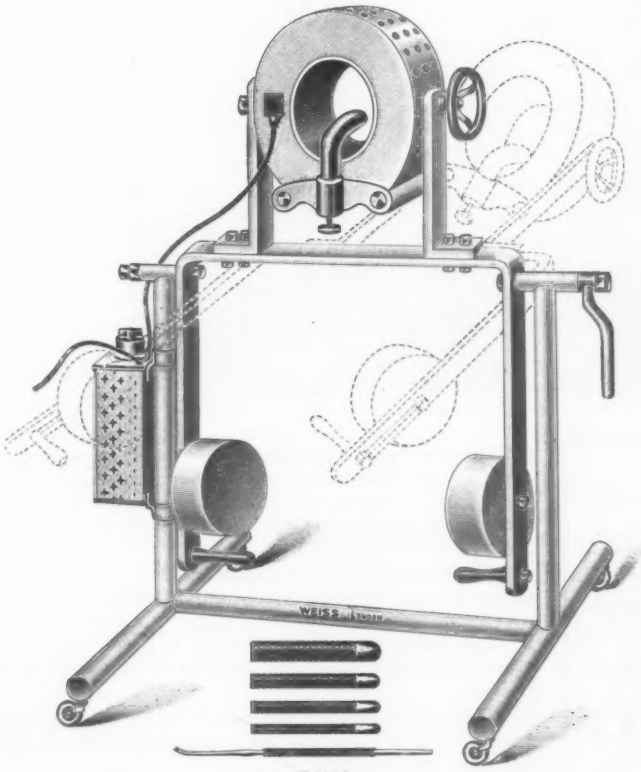

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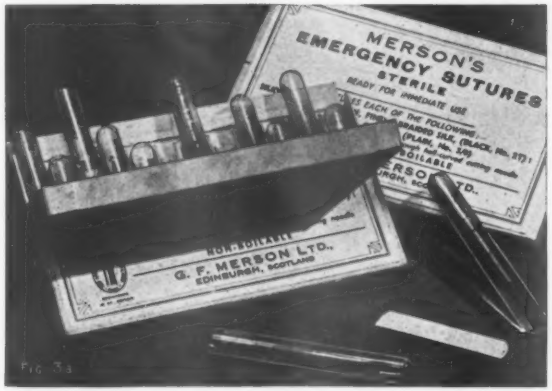

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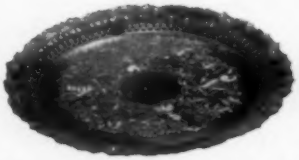

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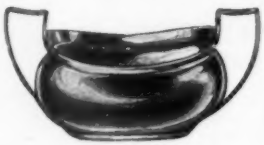

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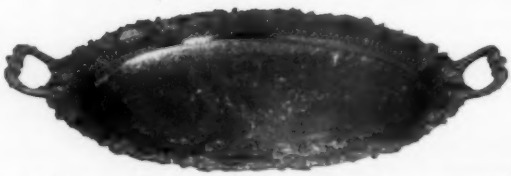

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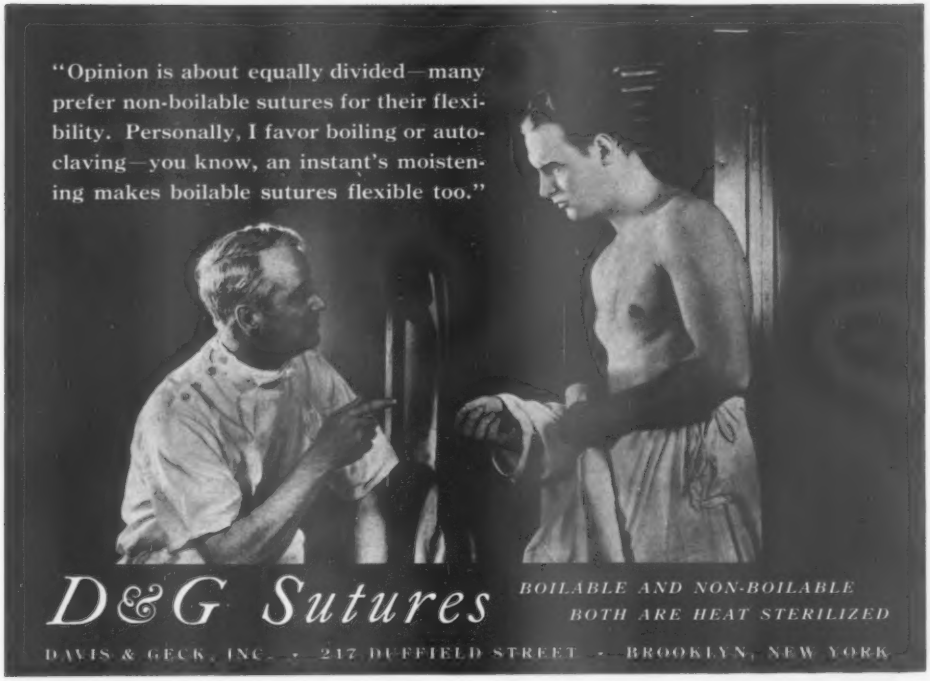
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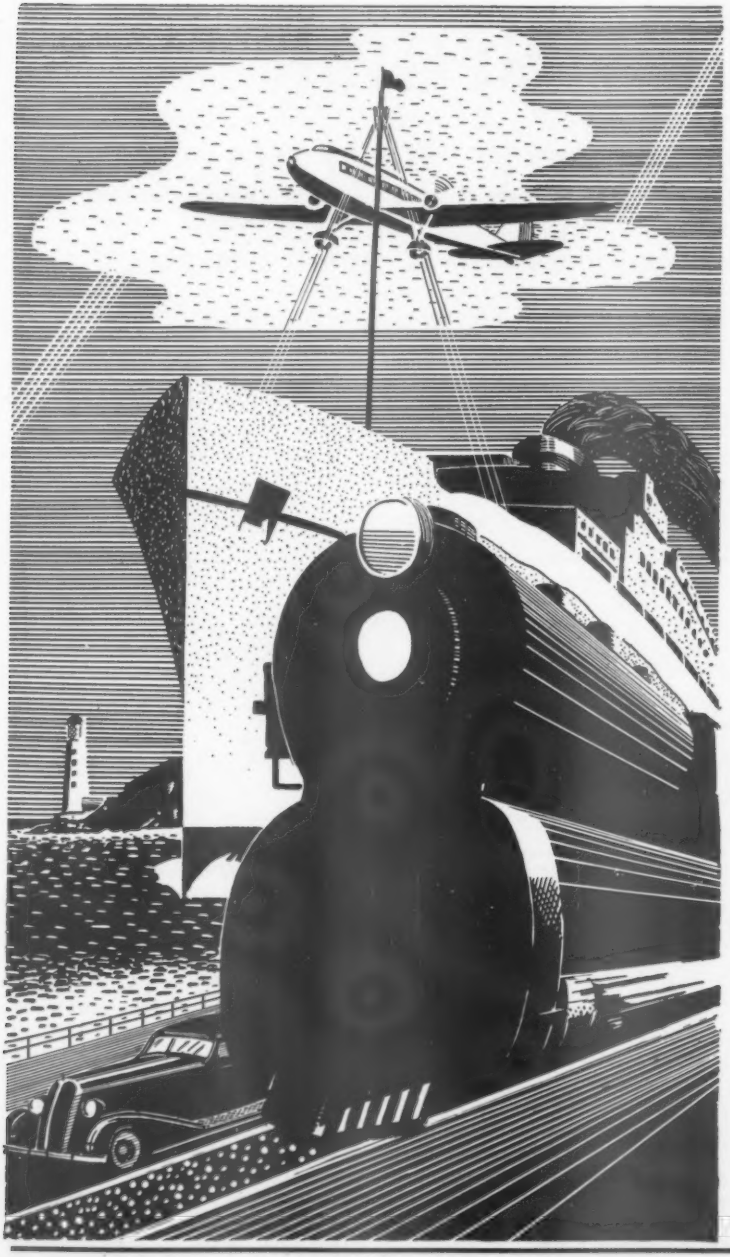

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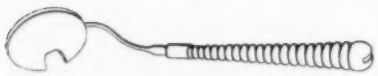

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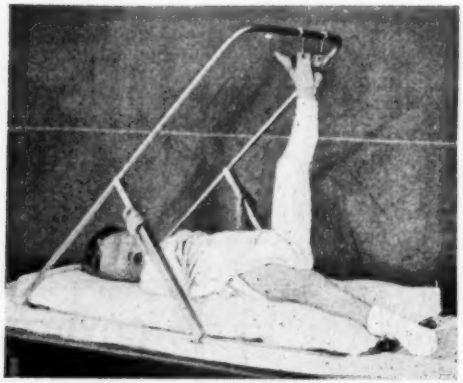

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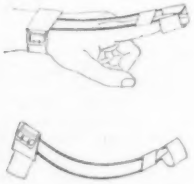

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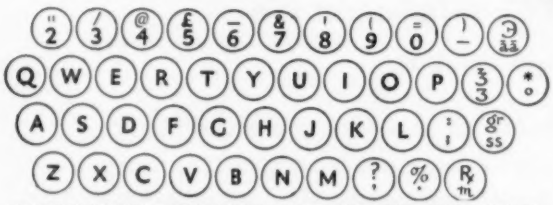

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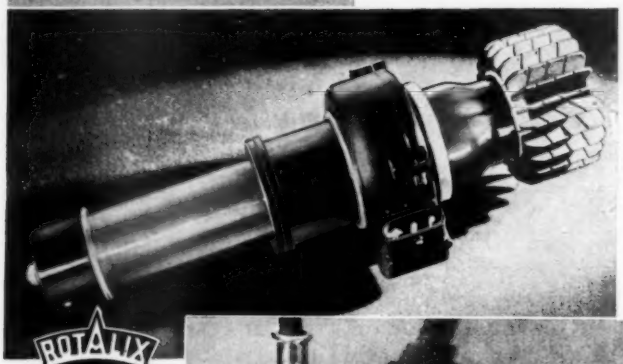

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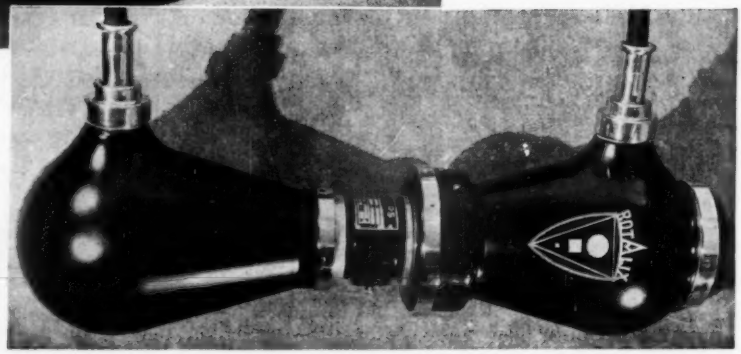

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The Australian and New Zealand Journal of Surgery

APRIL, 1938.

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VOL. VII. APRIL, 1938. No. 4.

VARICOSE VEINS AND THEIR COMPLICATIONS.

By DOUGLAS ROBB,
Auckland.

ANATOMY AND PHYSIOLOGY OF VEINS OF LOWER LIMB.

The Superficial System.

THE internal saphenous vein runs a well-known course up the inner aspect of leg and thigh from its beginnings in the inner part of the dorsal venous arch of the foot, until it terminates by turning deeply over the edge of the falciform thickening of the deep fascia, and emptying into the femoral vein. Three tributaries join it near its termination, the superficial external pudic, the superficial epigastric and the superficial circumflex iliac vein. Tributaries from the anterior and lateral aspects of the leg and thigh join it somewhat irregularly.

The external saphenous vein commences in the outer part of the dorsal arch, ascends behind the external malleolus, up the centre of the calf, to pierce the deep fascia and to terminate in the popliteal vein.

Communications between these two veins occur regularly on the postero-medial aspect of the leg, and occasionally on the same aspect of the thigh.

The Deep System.

The anterior and posterior tibial veins run as *venæ comites* to the corresponding arteries, communicating with one another at one or two places, and unite where the anterior veins pierce the interosseous membrane at the lower end of the popliteal space. The popliteal vein, thus formed, occupies the popliteal space, receives as tributary the external saphenous vein, and ends by penetrating the hiatus in the *adductor magnus* tendon. The femoral vein continues from this point to the level of Poupart's ligament, where it becomes the external iliac vein. Besides the internal saphenous, it receives another large tributary near its termination, the deep femoral vein.

Communications between the Superficial and Deep Systems.

In addition to the terminations of internal and external saphenous veins, which morphologically come under this heading, there are important perforating veins.

In relation to the internal saphenous vein, long narrow communications with the femoral occur in the middle of the thigh. In the leg, at the junction of upper and middle thirds, a wide channel passing close to the posterior border of the tibia to the posterior tibial vein is common, and of importance in relation to varicosity. Sometimes the hole which this vein makes in the deep fascia is palpable with the finger tip. At the junction of middle and lower thirds a communication runs to the posterior tibial from the internal or from the loop between the internal and external saphenous veins. Between these two a third perforating vein is often present.

In relation to the external saphenous vein in the upper two-thirds of the calf several indirect perforating veins connect the external saphenous with the posterior tibial vein, via the large muscular veins in the gastrocnemius and soleus muscles. In the lower third small direct perforating veins join the posterior tibial, either from the external, or from a loop between the external and internal saphenous veins.

The Valves.

At all points where the superficial system and the perforating veins terminate in the deep system, valves preventing reflux are present, and are capable of withstanding a very considerable pressure. Blood can flow only from the superficial to the deep system. The muscular tributaries entering the deep system are also all adequately provided with valves which prevent reflux. There are parietal valves in both external and internal saphenous veins, but these do not stand much pressure, and rapidly (particularly in the case of the internal) give way when the strong terminal valve becomes leaky.

The anterior and posterior tibial veins are throughout their course closely provided with valves which prevent downflow. The popliteal vein has a valve at its beginning and opposite the knee joint. The femoral vein has a competent valve at its termination, the femoral valve, and also below the entrance to the deep femoral vein there is found the deep femoral valve. The femoral valve is often the first competent valve between the heart and the limb, though sometimes there is a valve in the external iliac, and occasionally one in the common iliac vein.

Normal Venous Return from the Lower Limb.

Consideration of our erect posture and of the various physiological activities which raise the abdominal pressure through muscular action, makes the successful return of blood to the heart from the lower limbs

in all circumstances appear to be no mean feat. The forces responsible are:

1. Abdomino-thoracic pressure and suction, whereby during inspiration blood is sucked towards the heart by decrease of pressure in the thorax and increase in the abdomen. The iliac veins are thus emptied, particularly when the femoral valve is competent. During expiration the initial lowering of abdominal pressure helps blood to ascend from the limbs to the iliac veins. When violent straining with a fixed diaphragm occurs, a considerable back pressure takes place along the limb veins, causing difficulty or impossibility of emptying them unless violent muscular contraction occurs in the limb.

2. Contraction and relaxation of limb muscles. Remembering the arrangement of valves, it is clear that muscular contraction drives blood from the muscular and perforating tributaries into the deep system, and the blood in the deep system is driven upwards with great force. Relaxation then causes blood to enter the deep system from capillaries and the superficial system, to be driven up by the next contraction. The deep system, therefore, relieves the superficial system during muscular activity, the valves appearing as an anti-gravity mechanism and also as a mechanism for protecting the superficial system from too great a pressure.

3. The *vis a tergo* or force with which blood enters venules from capillaries is always acting, and is believed to be sufficient to return blood against gravity as long as the abdominal pressure is not unduly raised.

That the vein walls act as a peripheral heart in aiding the circulation, though stressed by some, particularly French writers, is not established. In standing, when the most potent force, the second, is absent, the third plays the main part, the first helping by emptying the upper segments. When a person stands at attention, the superficial system feels the strain most of all, and in hot weather when the vein walls are relaxed, the situation rapidly becomes intolerable. Muscular contraction and relaxation, however, speedily bring relief by carrying off the blood from superficial veins by deep channels.

ÆTIOLOGY OF VARICOSE VEINS.

Historical.

Doubtless owing to its conspicuous situation and unpleasant appearance, lower limb varicosity has attracted attention from time immemorial. Speculation as to its cause has been frequent, and to this day the question cannot be regarded as completely answered.

Up to the beginning of the seventeenth century, varicosity was attributed to the "stasis of crass melancholy blood reacting by its excessive weight on the walls of the veins". This humoral theory and its modern successor, the weak wall theory, according to which the vein walls are weakened by some acquired toxin or by inherited tendency, have been the resort of those who felt that mechanical factors

did not provide sufficient explanation. Various fascial and tumorous obstruction have at times been called in to account for varicose veins, but have proved inadequate for all cases. Valves in the veins were first demonstrated by Fabricius in 1579, and were used by his pupil Harvey as part of his proof of the circulation of the blood. The importance of incompetent valves was first realized in relation to the terminal internal saphenous region by Brodie one hundred years ago, and more fully by Trendelenburg in 1891. In relation to the perforating veins, Briquet was first in the field more than one hundred years ago, followed by Verneuil (*circa* 1855), Delore (1894), and Jeannel (1910), but it was not until Turner Warwick's experiments were made in 1931 that it could be determined whether this valvular incompetence was primary or secondary to acquired weakness of walls.

The Experiments of Turner Warwick.—Twenty-eight young subjects, up to about eighteen years of age, including several below twelve, without any clinical sign of varicosity, were studied in the *post mortem* room by injection into different parts of the venous system of the lower limb at varying pressures of coloured fluids, and also of radio-opaque fluid observed by screening. The limbs were subsequently dissected and the state of valves was noted. The family history of some of these subjects in relation to varicosity was also obtained. The subjects examined fell into the following types: (i) Normal or congenitally non-varicose type. In 70% of cases rises in pressure in the deep veins could not be transmitted to superficial veins, the valvular protection being adequate. (ii) Congenitally varicose internal saphenous type. In three of the twenty-eight subjects the valvular arrangements were inadequate to protect the superficial veins from increase in pressure in the deep veins, and the former readily filled by way of the perforating veins. Although there was no clinical sign of varicosis in these young subjects, their family history on the whole was strong in this respect. The commonest route of leakage was by the upper perforating vein in the calf.[3] (iii) Congenitally varicose external saphenous or calf muscle type. In two out of ten subjects reflux occurred through inadequate valves into the muscular tributaries, and thence into the external saphenous. In all, the valve at the termination of the external saphenous vein was competent. Both these cases had a positive family history for varicosis. (iv) Congenitally varicose deep vein type. An incompetent femoral valve reacts unfavourably on an incompetent internal saphenous vein, but has little effect on the deep system owing to the deep femoral and lower valves. In one of six subjects in this series the posterior tibial was found inadequately provided with valves over most of its course. This was a subject which came also under the preceding heading. Verneuil also described varicose posterior tibial veins.

Evidence from Four-Footed Animals and Native Races.

Many pathological conditions in man, such as postural deformities, ptosis of abdominal organs and certain gynæcological conditions appear

to be related to his singular erect posture. These conditions are often rendered more troublesome when a civilized way of life prevails, involving poor muscle tone, over-eating and less contact with sun, wind and sea. Such evidence as I have been able to collect with reference to quadrupeds and native races tends to place varicose veins amongst the conditions related to the erect posture.

In the horse the venous system of the limbs is similar to that of man, and there are numerous valves protecting the superficial system (Professor F. Wood Jones). In veterinary practice, Dr. Hopkirk, of Wellington, and Dr. Ring and Mr. Lyons, of Auckland, assure me that varicose veins are rarely, if ever, seen. Dollar (1930) states that varicocele is not common in animals, though well-marked cases have been found in the horse and ox. Varicose veins are likewise not common in veterinary patients, but are seen in the radial, cephalic, metatarsal, mammary (cow) and scrotal veins. The distended metatarsal vein in the horse is known as "blood spavin". Varicose ulcers are extremely rare. Stang and Wirth (1928) refer to the "blood spavin" as "fairly frequent". Hæmorrhoids occur most often in dogs, but isolated cases in cattle and horses have been observed. Varicocele is occasionally found in old stud stallions and in hounds. Barrier (1927) put the question to teaching and practising veterinary surgeons: "Do varicose veins exist in animals?" He referred to their extreme rarity in animals coming to dissection, and wondered whether this should be attributed to their relatively shorter lives. His inquiry brought forth "two cases of varicose veins in the horse" from Marcenac, an army veterinary officer. One case concerned an arterio-venous aneurysm the size of an orange on the right external iliac artery and vein. The second was one of varicose veins affecting the metacarpal veins of both forelimbs, attributable in his opinion to too early and too violent exercise, together with a congenital tendency of the vein walls to stretch. He quoted Floire to the effect that the metacarpal veins are sometimes varicose in the cow.

As regards native races, I have obtained information from Dr. S. M. Lambert (Fiji and most of the Pacific Islands), Dr. E. G. Sayers (Auckland and Solomon Islands), Dr. H. B. Turbott (Health Department, Hamilton, with reference to Maoris, Chinese and Samoans), Dr. J. S. Armstrong (Taupo, New Zealand, Samoa, India *et cetera*), Professor F. Wood Jones, Dr. G. M. Smith, North Auckland, and others who have worked amongst natives in New Zealand and elsewhere. Their reports, with but minor differences, were to the effect that varicose veins were seen in all native races. Where they lived under their natural conditions of easy life, squatting or lying down when tired, and having a diet which produced soft bulky motions passed in the squatting position, varicose veins (and hæmorrhoids) were not common, and gave little trouble. When they were observed living in a European style, as to diet and with fixed hours of labour, varicose veins and hæmorrhoids were much more common, extensive and troublesome. Coolies in South China, for example, carrying enormous loads, showed

them much more frequently and severely than Chinese in an easier way of life. Samoans, who mostly preserve their easy ways, do not show varicosity often. It would be interesting to test some of their adults who do not exhibit varicosity in the way that Turner Warwick tested his adolescents to determine whether their valves were fully competent or not. Natives in many countries, when they begin to be troubled in this way, either return or are sent back to their villages and their normal conditions, after which little is heard of their veins. That is why varicose veins and hæmorrhoids do not figure prominently in native hospital reports. Similar evidence comes from A. W. Burton (1934), who examined 33,308 male natives of between eighteen and forty-five years of age for work in the Transvaal mines. They came from the Xosa and Fingo tribes of the Bantu people, and were of good physique and happy-go-lucky temperament. They had lived in simple style in the open air, free from care and worry. Nineteen persons were rejected for severe varicose veins, affecting chiefly the internal saphenous and showing "defective action of the valves". This incidence is admittedly not high, but it occurred in more or less picked men who had hitherto lived under their natural conditions. It would be interesting to know how many of those accepted subsequently developed varices while at work in the mines. Only one case of varicocele was seen in the whole group. M. G. Cartoux (1936), on the other hand, states that varices, varicules, supramalleolar cyanosis, œdema of venous origin and varicose ulcer and eczema are almost unknown amongst the Tonkinese. Varicocele was found occasionally, and hæmorrhoids more often, but less than in Europeans. He follows Delater and other French writers in ascribing a large share in all varicose diseases to endocrine imbalance, which is rare in Tonkin.

In summary, therefore, it may be said that varicose veins do not occur in four-footed animals, and that they do occur in all native races, being more frequent and troublesome when the native lives under civilized conditions.

Summary of Evidence on Ætiology.

Putting together the material from the three preceding sections, we may present the causes of varicose veins as follows:

1. The prime cause is the assumption by *Homo sapiens* of the erect posture.

2. The chief mechanism is inadequate protection afforded to the superficial veins by the valves in the perforating veins, at the terminations of both saphenous veins and occasionally in the deep veins. These valves are present with some degree of efficiency in all human beings, and in the majority are sufficient to prevent the development of varicosity, even under the adverse circumstances of civilized life. In individuals who develop varicose veins, certain of the valves are absent or deficient from birth. Such defects are hereditary, often the identical valve or valves being deficient in a mother and daughter. Under favourable

conditions varicosity may not appear in persons with deficient valves. For example, an individual born with deficient valves rarely exhibits varicosity until after adolescence, because in the period of growth occupations involving prolonged standing are few, the muscles are constantly active and in good tone, the veins are smaller and have greater elasticity and the hydrostatic pressure, owing to smaller stature, is always less. The presence of one or more exciting factors, enumerated later, however, rapidly produces visible varices in those with deficient valves, and even at times in those with normal valves. The importance of valvular deficiency in causing varicose veins is demonstrated repeatedly during injection treatment, which obliterates the dilated veins. Recurrence of varicosity about the same site is regularly observed, because as a rule the leaky valve and perforating vein remain untouched by the injection. Whether or not the human race is gradually adapting itself better to the erect posture, I cannot say. It would be necessary to ascertain the incidence of varicose veins year by year over a long period, and also to discover whether valvular defects were gradually bred out of affected families or not.

3. Exciting factors are: lack of tone of muscles; overwork; convalescence after illness; occupations involving prolonged standing; any condition producing weakness of vein walls, such as pregnancy, in which some general or metabolic factor is at work, the varicosity developing before the uterus is large enough to obstruct mechanically the iliac veins; any mechanical obstruction to the venous return, such as pelvic tumour, and thrombosis of deep veins of limb (aponeurotic obstructions are not established as exciting factors); abdominal straining over a long period, from such conditions as constipation and enlargement of the prostate.

Circulation in Varicose Veins.

It is sometimes stated that the circulation in varicose veins is always downwards, and that this is one of the reasons why the thrombi produced by sclerosing injections never travel towards the heart and cause embolism. In the presence of an incompetent valve at the termination of the internal saphenous vein, a downrush of blood can be seen when the erect posture is assumed or pressure made over the termination of the empty vein is released while the subject is standing. But when circumstances have reached a state of equilibrium, and there is no unusual abdominal straining, the circulation in capillary loops has been found by Warwick (1931) to be the same when the subject is in both the erect and horizontal positions, though it is more sluggish in the former. The return circulation is thus maintained in both superficial varicose and deep veins in an upward direction by the *vis a tergo* aided by abdomino-thoracic suction, though at a slower rate than normal owing to the dilated condition of the veins. When a patient with no femoral valve and an incompetent saphenous system, however, engages in arduous efforts in the standing position (as in washing clothes), the

abdominal contractions so regularly stop, if not reverse, the return flow that the trophic changes of eczema and ulcer in the skin of the leg are favoured. In varicose veins of the leg without superior reflux, the circulation travels sluggishly towards the deep veins instead of freely, for muscular contraction, in addition to emptying the deep veins upwards, throws some of the blood back along incompetent channels to the varicose superficial veins, leaving more to be dealt with by the next muscular cycle.

CLINICAL TESTS FOR VARICOSE VEINS.

The tests applicable to a patient with varicose veins are as follow:

General Tests.

A general appraisement of the patient is always of interest and often of value. The family history, previous personal history particularly with reference to phlebitis, "white leg" and other circulatory troubles, and observation of the general build should be considered. In patients with major dgrees of varicosis, an ill-toned physique is often noted, and associated conditions, such as varicocele, hæmorrhoids, hernia, flat foot and other postural deformities are very commonly present. The examination should include the whole of the trunk as well as both lower limbs.

Impulse on Coughing.

Impulse on coughing along the distended veins above and/or below the knee indicates free communication with abdominal veins, and probably incompetence of the femoral valve and terminal and parietal valves of the internal saphenous vein. This test should be carried out with the patient's knees braced back and feet apart.

Trendelenburg's Test.

Trendelenburg's test is carried out by causing the patient to lie down and elevate the limb. With the surface veins empty, the termination of the internal saphenous vein is compressed and held while the patient rises to the erect posture. A positive result, signifying incompetence of terminal and parietal valves of the internal saphenous vein, is recorded when the veins fill with a downward rush of blood on release of the pressure.

Filling Time Test for Competence of Perforating Veins.

The filling time test for competence of the perforating veins is carried out as in Trendelenburg's test, except that the pressure is maintained. The empty veins in the leg are watched, and if they fill readily (under forty seconds is a time given) the perforating system is incompetent; if they fill slowly (in more than sixty seconds) it is competent.

Rubber Band Tests for Competence of Perforating Veins.

1. Rubber bands are applied to the leg above and below the varicose veins when they are empty, with the patient in the lying position. When the patient stands, the veins fill rapidly if there is a large incompetent vein within the segment tested. A small vein allows only slow filling.

2. A particular perforating vein suspected of incompetence may be similarly tested with a band above and below, applied first with the varicose veins full and, secondly, empty. The patient walks up and down the room. If the veins are now empty, the perforating vein is competent. If the empty veins have filled it is incompetent. These two tests do not always give definite information.

Tests for Patency of Deep Veins.

On clinical grounds, de Takats (1932) states that the deep veins may be considered adequate if the patient is up and about and there is no swelling of the ankles or legs.

Perthes's test is quoted by de Takats (1932). A cuff is thrown round the thigh, with the patient in the erect posture, so as to compress the saphenous vein, and the patient walks to and fro, or flexes and extends the knee ten times. The dilated veins diminish if the deep veins are patent. The cuff is now released, and the veins fill from the saphenous vein. If there is no appreciable diminution on walking, some obstruction is present in the deep veins.

Warwick's rubber band test is made in the following way. If it is suspected that the superficial varix between the upper and lower main perforating veins is compensating for a deficiency in the same segment of deep veins, the two-band test is carried out as explained above. If the full veins become empty, the deep veins must be patent.

Most writers agree that it is sometimes difficult to be certain of the condition of the deep veins, and that the latter two tests are apt to give equivocal results. A careful history is of great value. The presence of œdema, under average conditions of activity, and in the absence of any general cause, is taken to signify permanent damage to the return circulation by veins and lymphatics.

Tests for Coexisting Arterial Disease.

In treating varicose veins it is important to bear in mind the possibility of coexisting arterial disease, for the impoverished tissues react badly to injections. Indeed, it is said that varicosity or phlebitis may be the first sign of Buerger's disease. Warning features are muscular cramps, feebleness of pulse in the main vessels at the level of the ankle, clamminess and blueness of the leg, and trophic changes about the toes and soles. In case of doubt, more accurate observations may be made with Pachon's oscillometer, or by the skin response to histamine (Lewis) as advised by Warwick (1931) and de Takats (1930).

THE CLINICAL TYPES OF VARICOSE VEINS.

Varicose Veins Dependent on Valvular Deficiencies.

When the terminal and parietal valves of internal saphenous veins are deficient, the main trunk in the thigh and leg is usually, though not always, greatly dilated and filled constantly by the superior reflux. If the femoral valve also leaks, as it often does, the condition is worse, and there is an uninterrupted column of blood from heart to foot. A feeling of weight in the limb is a constant symptom. Walking does not relieve this feeling, as blood removed by the perforating veins is at once replaced from above. This symptom is immediately relieved by lying down. It is surprising how accustomed patients become to this feeling of weight. Sometimes it causes them little inconvenience; nevertheless they appreciate greatly the relief afforded by the Trendelenburg operation.

When the valves of the perforating veins in the leg are deficient, varicosity appears in the internal or external saphenous veins opposite the valves affected. Varicosity at the junction of upper and middle thirds of the leg is perhaps the commonest, with varicosity a third lower next in order. Sometimes both a mother and daughter will exhibit varices in similar positions. In varicosity of the external saphenous vein either direct or indirect perforating veins may be at fault, and varicosity may appear first in the surface veins of the calf, or in them subsequent to dilatation of the muscular veins. It may occur secondarily to the internal saphenous vein by way of communications. The terminal valve of the external saphenous vein may be leaky, giving rise to clusters in the neighbourhood of the popliteal space. In all these cases the feeling of weight is not marked, but its place is taken by a local aching. This is relieved by walking or other exercise, as the engorged veins are thereby partly emptied. It is this group that is especially suitable for treatment by injection.

Any combination of valvular deficiencies may be present, and the resulting varicosity may reach any size. When a number of leaky perforating veins in the leg are combined with a leaky terminal valve in the internal saphenous vein, the whole superficial system becomes extensively varicose.

Deficiency in valves of deep veins is important more from its secondary effects. In deficiency of the femoral valve the strain is thrown on the internal saphenous vein, as the valves in the deep veins of the thigh are usually competent. In the posterior tibial the condition is recognized clinically through the stretching of the vein walls and the valves guarding the perforating veins. Such a leg requires permanent external support, as the deficiency renders muscular contraction futile as a force in raising the blood.

Varicules in the skin may be mentioned here, though they bear no apparent relation to valvular deficiencies. They occur as patches of fine bluish lines about the thighs and legs, usually in women. They are unsightly, but do not ordinarily require treatment, though Schmier

(1930) states that they can be made to thrombose and to disappear by subcutaneous transfixion and scarification with the point of a fine needle.

Varicose Veins Dependent on Obstruction to Deep Veins.

In the abdomen, blockage of the inferior *vena cava*, common or external iliac veins produces the well-known clinical picture of anastomosis between Scarpa's triangle and the veins of abdominal wall and axilla, or the anastomosis extends across the middle line to the opposite groin and/or axilla, as the case may be. It is important to bear these conditions in mind, as the whole superficial system in the lower limb or limbs may be varicose as a result of one of them. Unless the examination of the patient includes the trunk, a vain and possibly harmful course of injections in leg and thigh may be undertaken.

In the thigh and/or leg the superficial system again may be doing duty for the deep system, and should not be lightly injected in whole or in part.

In both these groups the appearance of the leg and ankle usually suggests the deeper disorder. Œdema and pallor, with thickening of subcutaneous tissues and trophic disorders of the skin, are almost always present.

TREATMENT OF VARICOSE VEINS.

Our hypothesis is that varicose veins result from the action of one or more exciting factors in an individual usually with congenital deficiency in certain of the valves of his veins, or occasionally with normal valves. General and conservative measures are therefore of value to cure the condition in some cases, to relieve it when other measures cannot be applied, and to postpone recurrence and complications when the valvular defect is intractable. Sclerosis by injection is the method applied in most cases, whatever their anatomical type, except that when there is a positive response to the Trendelenburg test special measures must be taken, usually operation, in addition to injection.

General and Conservative Measures.

The general health must be looked to, and too much standing avoided. Toxic and obstructive conditions should be searched for and, if possible, relieved. To the veins themselves external support may be given by advising a boot instead of a shoe, or by bandaging with a crêpe bandage. Bandaging is often sufficient to tide a patient over pregnancy or convalescence from illness (to avoid phlebitis), and may be made more effective for short periods by use of an Unna's paste cast, or "Elastoplast" strapping. The elastic stocking is a useful measure when more permanent support is required; for example, a limb with deep venous obstruction.

Sclerosis by Injection.

Sclerosis by injection, a relatively new procedure, has in ten or twelve years established itself as a safe and easy method of obliterating

varicose veins. In some respects it is too easy, for its unskilful or ill-considered application has led to serious trouble. None of the points discussed in this lengthy paper is irrelevant to the matter, and even the simplest case for injection requires thought beforehand, care in execution and knowledge, if an accurate prognosis is to be given.

I use the quinine and urethane solution as a routine, having found it the most generally reliable in causing sclerosis, painless during injection, and without common general ill-effects. It is not invariably effective, sometimes failing completely, at other times causing the "tarry reaction" discussed later. Severe reactions to the quinine occur occasionally, in women as a rule, and may be anticipated by questioning, and giving a small initial dose. The risk from the quinine in pregnancy is overrated, but if it is desirable to give injections at all, I use another solution. The quinine solution causes pain if deposited outside the vein and usually necrosis of tissue and ulceration. I have used most of the other solutions advised, sodium morrhuate, lithium salicylate, sodium salicylate, glucose and sodium chloride, but for various reasons, chiefly unreliability in causing sclerosis, keep some of them as alternatives only for use when quinine fails or its use is inadvisable.

As to technique, I aim at injecting the solution into an empty or almost empty vein, and have the patient lying flat, sitting with leg horizontal or vertical, or standing, to attain this condition. The vein must, of course, be sufficiently distended to allow the placing of the needle correctly, but only occasionally is a tourniquet required. After the injection is given, the puncture is compressed with a swab for two or three minutes and a pad and bandage are put on for one or two days. Both these points are important in order to diminish the chance of leakage of injection fluid through the needle hole in the vein wall. Pain during injection suggests leakage of fluid around the vein, and calls for immediate cessation. Indeed, if the needle is not correctly placed on the first attempt, it is wise to try another place, or to postpone the injection. After the first small dose of the quinine solution (0·5 to 1·0 cubic centimetre), as much as four or five cubic centimetres may be given over two or three situations at one session. Sessions are best separated by a week, but the interval may be reduced to four or three days. Varices are usually injected from the ankle region upwards, the operator stopping at the lower third of the thigh. The trunk of the internal saphenous vein in the thigh is considered later. Sometimes a period of rest or "Elastoplast" bandaging is necessary or desirable to reduce swelling and to demonstrate the veins more clearly before injection.

Sometimes a vein will not become sclerosed after repeated injections with different fluids. Those with very thin surrounding and covering tissues are apt to behave in this way. The only way to get rid of such a vein if it is very unsightly is by a local excision. At times an incomplete reaction takes place, when the walls are partially affected and a segment of vein retains thick tarry blood. This is a troublesome

condition which causes aching and sometimes throbbing; it is usually apparent within a week of injection, though sometimes not for two or three weeks. I am at a loss to account for this behaviour. Relief is tardy, unless the tarry blood is removed by expression following puncture with a needle larger than the fine one used for the injection. Sometimes the segment cannot be emptied in this way, and is best treated by a local excision. Usually obliteration takes place after evacuation of the tarry blood, but sometimes a further injection must be given. Another unpleasant effect is a transient ascending phlebitis, much beyond the usual limits of reaction to the injection. A group of veins is injected in the ordinary way below the knee, and two or three days later the patient presents a red angry saphena, inflamed perhaps up to Scarpa's triangle. Fortunately this condition subsides without ill-effect and leaves a well thrombosed trunk in three or four days, if the thigh is well padded and strapped as described in the treatment of acute superficial phlebitis. It is probably due to a chronic or latent phlebitis (see later), unsuspected or unrecognized when the injection is made.

Contraindications to injection therapy are advanced general conditions of ill-health, recent phlebitis, the presence of arterial deficiency in the limb concerned, and inadequacy of the deep venous system. The last mentioned is not an absolute contraindication, for specially prominent or troublesome veins may be obliterated safely by small doses of fluid, but, on the whole, such a limb is best treated conservatively.

An accurate prognosis as to recurrence after injection can usually be given, if the time taken for the original varices to develop and the type are correctly assessed. In a simple cluster in the leg, the injection obliterates veins distended as a result of a leaky valve. The leaky valve itself remains untouched.

Operation.

The main indication is the presence of a clearly positive result to the Trendelenburg test. The downward reflux minimizes and sometimes nullifies the effects of injection in thigh and leg. When injections have apparently succeeded, the limb is usually found to be as badly affected as ever in a few months; moreover the feeling of weight is unrelieved. The presence of obstruction to deep veins in abdomen and limb is excluded before operation is considered.

The safest, most certain and final way of checking the reflux is by Trendelenburg's operation, easily performed under local anæsthesia. The upper four inches of the internal saphenous vein are excised, and the stump is firmly ligated flush with the side of the femoral vein. The three main and any other tributaries are ligated at a little distance. The deficient valve is thus removed, the column is effectually broken, no pocket is left to harbour a clot, and the possibility of lateral anastomosis via the three tributaries is removed. Remaining varices, often associated with leaky valves in the calf, are injected. When these valves are leaky, the corresponding varices are likely to recur and require

reinjection, but, of course, more slowly and less extensively than when subjected to the superior reflux.

Excellent results have followed this operation in my hands. I have performed it for 33 (21 male, 12 female) out of 548 patients suffering from varicose veins. A considerable number of patients in addition gave a positive result to the Trendelenburg test, but for various reasons were not operated upon. I am aware of one recurrence, for which I am at a loss to account, and have not been able to see; and one patient during convalescence had symptoms suggesting a minor pulmonary embolus. The last-mentioned risk is minimized by free movements of the limb in bed, beginning the day after operation. The operation requires admission to hospital, and two or three weeks off work. Sometimes a patient cannot or will not face this, and insists on the best being done by injections only. In such circumstances I have injected the trunk in the thigh, either piecemeal from below upwards, guarding the termination by thumb pressure for five minutes after each injection, or by injecting four cubic centimetres into the upper end, releasing the reflux, and driving the solution downwards. I have found it difficult to achieve a satisfactory sclerosis of the trunk by injection, and regard it as impossible to secure obliteration of the upper end where the leaky valve is situated and where obliteration is most needed. For these reasons I undertake injection only as a procedure that is second in value. I must confess, however, to being confronted with a patient whom I had seen seven years before, when I had advised operation. He had gone elsewhere and had the trunk of his vein injected. In answer to my follow-up inquiry, he showed an excellent leg, with the trunk quite obliterated, and no recurrence at all.

I am aware of the practice of "ambulatory ligation" of the trunk of the saphenous vein, largely followed in the United States of America (for example, de Takats, 1930), as an alternative to the operation. I have not used it because I cannot imagine the ligation being performed high enough to lie above the leaky valve without open operation. There will therefore be both the valve and a blind pouch of vein above the ligature. The former must lead to recurrence of the reflux, the latter to a risk of embolism from breaking off of a clot. De Takats (1930) claims to have completely avoided embolism through ambulatory treatment, and contrasts his experience with another series showing 0·7% of fatal embolisms after use of the same type of ligature followed by rest in bed. Another objection is that the three large tributaries still remain, and are available for anastomosis to some extent whether the ligature is placed above them or below.

Other operative procedures in varicose veins can be dismissed quickly. The excision of a segment or cluster resistant to injection and the possible ligation of an inveterately leaky perforating vein in the calf, as suggested by Turner Warwick, are amongst the few required.

COMPLICATIONS AND THEIR MANAGEMENT.

Hæmorrhage.

Hæmorrhage results from atrophy of skin over a prominent varix, and copious hæmorrhage takes place until the patient lies down and elevates the limb, or a firm pad and bandage are secured over the leak. The nearby veins should be injected at once, and if this is done the necrotic patch quickly heals. When the bleeding takes place from the bed of an ulcer, the usual methods for cure of an ulcer are undertaken, a start being made with an "Elastoplast" bandage.

Eczema and Ulcer.

I regard eczema and ulcer in a varicose limb, when general states such as diabetes are excluded, as the earlier and later results of malnutrition of the skin due to prolonged contact with venous blood. Frequently a particular vein will point at its lower end to a patch of eczema or to an ulcer. In the œdematous tissues at the edge of an ulcer invisible cavernous varices may be felt, and the veins are very obvious after the œdema is removed by rest or strapping. The ill-nourished hairless skin of a varicose leg is very susceptible to trauma. Pain in an ulcer seems to depend on the exposure of nerve terminals, though in old and extensive ulcers it is sometimes due to a subjacent periostitis.

The presence of either eczema or ulcer is an indication for immediate improvement of the circulation. Treatment of the veins by injection and/or operation should be combined with external support to reduce congestion and œdema. At the first session one or two cubic centimetres of solution may be injected in veins a few centimetres above the ulcer, and the whole limb, from toes to knee, may be bound firmly in "Elastoplast". The latter may be applied directly over the surface of the ulcer after it has been cleansed, but I usually interpose a patch of lint a little bigger than the ulcer, coated with cod liver oil and "Vaseline". Immediate relief is felt, and remarkable improvement in the ulcer is seen when the "Elastoplast" is removed in seven, ten or fourteen days' time. More veins are then injected and fresh "Elastoplast" is applied, and so on until the ulcer is healed. The ambulatory nature of the treatment is good both locally and generally. Seepage of discharge through the "Elastoplast" is unpleasant, but usually minimal after the first change. Sometimes "Elastoplast" cannot be used. Pain in the ulcer itself, in adjacent inflamed veins or in swollen tissues around the ulcer may make it intolerable. The painful ulcer may be only a tiny one near one or other malleolus, and yet prevent the use of "Elastoplast". Eczema and excoriation of skin may likewise prevent its use, either when the patient is first seen or after the first or second bandages have been removed. In these cases I find an Unna's paste cast an admirable substitute, with or without a dressing of oil and "Vaseline" underneath. The Unna's cast is best applied after rest if œdema is present, and is soothing and

effective. In one or two cases of painful ulcer and periphlebitic indurations (see later) I have had to resort to a light plaster of Paris case, from toes to below knee, applied after œdema has been reduced. This is very effective in stopping the pain at once and promoting healing, and may be left on for three weeks or more at a time. The help to healing of ulcers by treatment of veins and elastic support has rendered unnecessary procedures involving skin grafting in all except the most extensive cases.

Superficial Periphlebitis.

Superficial periphlebitis is usually seen in older women with moderate rather than extensive varices. It is usually situated in the lower half of the leg, and takes the form of patches of stiff induration in the skin and subcutaneous tissues. The skin is often shiny and dull red in colour, and the whole area is painful. A varicose vein may be seen or felt to run through or close by the patch. Sometimes recognizable varices are present only at a distance. This condition is not an ordinary phlebitis, as it extends far beyond the walls of the veins, which often feel quite thin. There is not as a rule much swelling in or about the patches, and little in the leg or foot as a whole. Ulcer is not commonly associated with periphlebitis. "Elastoplast" binding may be tried, but is usually ill-tolerated. All signs of inflammation are removed by immobilization for six to twelve weeks of the foot and leg in a series of light plaster of Paris cases in which the patient can walk. The veins are then fit for injection, rather smaller doses than usual being used at each session.

Thrombosis and Phlebitis in Superficial Veins.

Payne (1936) has discussed the meaning which should be attached to the terms thrombosis and phlebitis in superficial veins; and I follow him in applying the term phlebitis to any of the conditions in veins which lead to thrombosis. At one end of the scale we have a quiet thrombosis wherein bacterial inflammation plays little or no part; at the other a bacterial inflammation with the formation of septic clots and emboli. An intermediate form is the common acutely thrombosed and inflamed vein, with mild fever and angry red overlying skin, which resolves completely without pus formation under appropriate treatment.

The treatment of these cases of acute phlebitis in superficial veins is very satisfactory. A thick pad of wool or "Sorbo" rubber is placed over the healthy vein above the inflamed area, and a flatter pad over the area itself. Firm strapping with "Elastoplast" over the pads from above down enables the patient to get about safely. Resolution, even in the presence of mild fever, takes place rapidly, and the vein does not as a rule become recanalized later. In the most extensive and spreading cases with greater fever, there is some difference of opinion as to the correct treatment. Dickson Wright (1936) confidently advises similar treatment, laying strips of "Sorbo" rubber along the inflamed veins and

bandaging very firmly with "Elastoplast". He states that the more severe the case, the more salutary the results. Keeping the patient up and about is essential to prevent embolism and deep thrombosis. Payne (1936), on the other hand, puts his patient to bed for a month, and then excises the inflamed mass of veins with any skin involved. When the local lesion is a septic one and has produced, or seems likely from the temperature chart and local signs to produce, pulmonary emboli, he advises immediate ligation of the saphenous vein at the sapheno-femoral junction, with or without turning out of septic clots. I have not had sufficient experience of severe cases to take sides definitely, but I incline to Dickson Wright's view. In only one case have I resorted to operation for fear of embolism. This was one of acute phlebitis in the whole of an enormous internal saphenous vein. Trendelenburg's operation, followed by excision of the whole mass, gave a good result.

The more chronic and latent forms of phlebitis are also of importance in relation to treatment. They may follow an attack of acute phlebitis, and may be masked while the patient is at rest or under treatment, only to cause pain and tenderness on the resumption of activity. There is a great tendency to recurrence of attacks in the same segment of vein or in different parts of the same limb. At other times a history of acute phlebitis even of mild character cannot be elicited, and the trouble may be suspected from such signs as old pigmentation, phleboliths and thickened tender walls in the veins. De Takats (1932) suggests the following provocative measures to elicit signs in a vein with latent phlebitis: gentle massage, heat, diathermy and needle puncture, with or without the injection of three minims of a 50% solution of glucose.

It is important to inquire for and search for evidence of previous acute phlebitis and latent phlebitis, for most authorities agree that it is unwise to inject incautiously the veins in such a patient. De Takats (1932) does not use injections until at least three months after an acute phlebitis, but I feel safer in following Payne (1936) in making the period six months. The risk lies in lighting up acute phlebitis elsewhere in the same limb, or even in the opposite limb, for the venous system seems to be a coordinated whole and is inclined to react often at a distance to fresh assaults. The risk is admittedly not such a serious one now that the ambulatory treatment of acute phlebitis is so effective. Indeed, some writers, for example, O'Neil (1931) and Lowenberg (1934), recommend immediate injection of healthy veins in the neighbourhood of acute or latent phlebitis as an aid to resolution by pressure. Nevertheless, I prefer to treat patients so affected conservatively by pressure and support only, or simply to observe them, for the arbitrary period of six months before resorting to injection. There is always the chance that phlebitis may be precipitated in a deep vein, which is more serious. The excision of a segment of vein which is troublesome from recurrent phlebitis, should be done only after a period of quiescence, and is rarely called for on account of the success of injections.

The subject of *thrombophlebitis migrans* may be touched on in connexion with recurrent phlebitis. This is a general disease of the venous system, with no special relation to varicose veins, except that the first attacks of phlebitis commonly occur in superficial varicose veins. Sooner or later thrombosis occurs in other veins in other parts of the body at different times. One patient of mine had thrombosis in both superficial and deep veins of leg, uterine, mesenteric, coronary, pulmonary, jugular and intracranial veins at different times. Obscure troubles in abdomen, thorax and brain in patients with recurrent phlebitis in limbs may be due to this disorder. Accounts with case records have been given by Moorhead and Abrahamson (1928), Ryle (1930), Hartfall and Armitage (1932) and others.

Thrombosis and Phlebitis in Deep Veins.

The subject of thrombosis and phlebitis in deep veins will be dealt with, not comprehensively, but mainly in its relation to varicosity. We have seen that surface veins may become varicose through doing duty for deep veins which have become blocked, and how to recognize the presence and situation of such a block has been discussed. The clinician should approach the treatment of these varices with caution, realizing that the limb is permanently damaged as to its return circulation, both venous and lymphatic, and that most of the symptoms are due to the resulting œdema and thickening of tissues. Injection therapy is not absolutely contraindicated by the presence of deep thrombosis, but it should be carried out slowly with small injections (0·5 to 1·0 cubic centimetre of quinine and urethane at a session), abandoned at once if a general swelling reaction is encountered, and limited to particular segments which are troublesome or adjacent to eczema or ulcer. For the most part, the varices are a minor factor in such a limb.

The treatment of a limb thus permanently damaged resolves itself into giving as much support and help to the circulation as possible, and reducing the time in which it is allowed to be swollen, for swelling leads to further thickening. Rest with the limb elevated, the use of a boot instead of a shoe, firm bandaging with crêpe bandages, and the periodical use of "Elastoplast" strapping are all helpful measures. Perhaps the most generally useful aid is a well-fitting elastic stocking, applied on rising from rest. A course of massage every few months may be advised. Ulcer in such a limb is very intractable, but may be healed by injection of veins near by and repeated application of Unna's casing or "Elastoplast". Recent accounts (for example, Murphy, 1937) of the treatment of swollen limbs after thrombophlebitis are encouraging. Iontophoresis with acetyl-β-methyl choline has given relief from pain and swelling, and favoured the healing of ulcers. In view of the intractable nature of the condition the method deserves further trial.

When a deep thrombosis occurs for the first time, rest in bed and elevation of the limb should be enjoined for six weeks, followed by gradual return to activity within the limits causing only slight swelling

and the use of support. Some permanent damage almost always results and with a view to diminishing this some writers, for example, de Takats (1933), have advised more active treatment at an earlier stage. Minor movements, such as sitting up and turning on the side, are allowed for ten days after the temperature and pulse are normal. Then stroking massage for five minutes a day is allowed for the next week. The foot is allowed to hang down during the third week, and the patient is allowed out of bed in the fourth week after the temperature and pulse are normal. Elastic support is begun at this stage and continued for three to six months.

Pulmonary embolus is the bugbear in these cases, but, as Payne (1936) points out, it is encouraging to remember that few, if any, patients coming to *post mortem* examination as a result of pulmonary embolism have shown signs of œdema of the lower limb during life. Many attempts have been made to find an effective prophylaxis against post-operative thrombosis and embolism. Free movements of the lower limbs in bed, commencing the day after operation, with bandaging of superficial varices, are well-tried and valuable measures. Treatment with thyreoid extract and thyroxin has been carefully studied (Cutting, 1931), but is considered of doubtful value, and sometimes undesirable on general grounds. Bankoff (1934) recommends the administration of one one-hundredth of a grain of atropine and one-quarter of a grain of ephetonin every other day for three doses, beginning on the fifth day after operation on his "thyroxin resistant" patients. This is a simple procedure, and would appear to warrant further trial. Similarly, the intramuscular injection of one cubic centimetre of calcium chlorate daily in the post-operative period would appear to be worthy of further extended trial. (Reference to this will be found in *The Practitioner,* Volume cxxxix, 1937, page 515.) Homans (1934) recommends ligation of the femoral vein, between the terminations of deep femoral and internal saphenous veins, in order to forestall pulmonary embolism in thrombosis of deep veins of leg or calf. This procedure also seems to me worthy of consideration, especially when the patient has a history of exacerbations of trouble deep in the leg, and when he has already survived one pulmonary embolus, and the signs in the leg persist. In some cases it will fail because the clot has already extended above the point to be ligated.

ACKNOWLEDGEMENTS.

It is a pleasure to record the help I have received in my study of varicose veins from Mr. Turner Warwick, of the Middlesex Hospital, not only through his book, but also through written and verbal communications. My grateful thanks are also due to those named in the section dealing with ætiology.

THE ÆTIOLOGY OF PHLYCTENULAR OPHTHALMIA.[1]

By HOWARD COVERDALE,
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My excuse for this short paper is that I have recently seen some severe cases of phlyctenular ophthalmia in Auckland in children in whom I should have considered unlikely subjects for the disease; that is, they seemed otherwise healthy, lived mostly in the open air, and may be presumed to have had a suitable diet.

It may with advantage be asked why we see so little phlyctenular ophthalmia in New Zealand, and it would be of interest to know how prevalent it is in Australia and to what it is generally attributed there. Perhaps it will be possible in time to find some common factor, and to this end observations made here may be of value.

I have no solution of my own to offer; I propose merely to state the problem as it appears to me in the light of such observations as I have made.

HISTORICAL.

A survey of some of the textbooks of the last three centuries reveals the fact that the disease was common during this period, although it passed under various names, and, until the nineteenth century, was not well described.

Bannister, in 1622, spoke of "phlyctenæ—hot little blisters" arising in the skins of the eye. Over a century later the textbook bearing the name of the notorious Sir William Read added nothing to Bannister's description, but Maitre-Jan, in 1740, noted the occurrence of phlyctens or pustules—at that time synonymous terms—on both cornea and conjunctiva. In the following year, de St. Yves gave a more satisfactory description of the conjunctival lesions, but did not include the corneal lesion, unless it is recognizable under the name of "humid ophthalmy", associated with ulcers, photophobia and pain. He noted the presence of scabs and pustules on the lips and face, and considered these to be due to discharge from the eyes.

The nineteenth century brought the name scrofulous ophthalmia. Antonio Scarpa, in 1818, said that no treatment had been discovered. Robert Hull, in 1840, was almost as pessimistic. He remarks:

> The great miseries and mischiefs of a scrofulous ophthalmy are chiefly witnessed in the children of the poor . . . . how long and intractable their affections are, the records of an eye infirmary demonstrate. How prone to occur again and again. What can mere medical agents effect for these miserable victims?

[1] Read at the annual meeting of the Royal Australasian College of Surgeons, Auckland, January, 1937.

Hull unhelpfully defined strumous ophthalmia as inflammation in a strumous subject.

Lawrence (1833) and Tyrrell (1840) used the terms strumous and scrofulous. They both stressed the excessive photophobia as being characteristic, and suggested a constitutional cause. The latter thought that the most important of such factors were skin and abdominal disorders, especially at the time of puberty. Counter-irritation was their chief local treatment.

The meaning of scrofulous is not well defined. The term still persists in reference to this disease, and Duane and De Schweinitz both use it freely. Hooper's dictionary of 1825 and Copland's of 1858 clearly intended scrofula and struma to mean tuberculosis, though, since Robert Koch, of Göttingen, did not discover the tubercle bacillus until 1882, enlarged cervical glands from infected tonsils or impetigo would probably have been called scrofula. That this was so is indicated by the "Cyclopædia of Practical Medicine" of 1834. The authors remark on the increased sensitiveness of the skin to irritants in strumous individuals and go on later to say:

> The absorbent glands, indeed, are the parts of the body which have long been regarded as the peculiar seats of scrofula, and their enlargement and induration are still considered as affording more unequivocal evidence in the living body of the presence of that disease than any other circumstance except the discharge of tuberculous matter.

The authors quote Beer as saying that, in Vienna, nine-tenths of all the cases of ophthalmia in children were of a scrofulous character, and Benedict, of Breslau, as estimating the proportion in that city as 95%.

This glance at the history of the disease during the last three centuries shows that it was associated with skin eruptions as early as 1741; that also it was not termed scrofulous until the nineteenth century, and then in a sense which embraced many skin eruptions and glandular swellings from almost any cause.

DISCUSSION.

I have made investigations of the cases under my care regarding age and sex, history of onset, economic conditions, tuberculosis, nose and throat, cervical glands, pediculosis, skin affection and refraction. These investigations have not disclosed any facts upon which a theory may be based with confidence.

Gibson, in 1918, carefully examined a series of 92 patients from the point of view of general health, and found that "52 were well developed and nourished, 28 fair and 12 decidedly below par".

Before discussing current theories of ætiology, I should mention that babies of less than one year old do not seem to be affected. Four to five years is the commonest age. I should also mention that institutional treatment, in hospital or sanatorium, usually effects a temporary cure.

Tuberculosis.—There is now almost general agreement that a constitutional factor is concerned in phlyctenular disease, and the view

most commonly held is that it is a manifestation of tuberculous toxæmia. It is not suggested that phlyctens are actual tubercles.

The evidence for this view rests on: (i) The supposed high percentage of the patients either suffering from tuberculosis or having a family history of tuberculosis. (ii) The high percentage of positive von Pirquet reactions. (iii) The experimental production of phlyctens with tuberculin.

Regarding the first of these three observations, it is difficult to comment on the findings in the absence of more detailed information than is usually given. Though not obvious in my cases, there does seem to be an unusually high proportion of patients in whom a tuberculous lesion can be found. This is, however, to be expected in the children of the very poor, and it is a little surprising that in 84 cases examined by him for transference to the County Ophthalmia School, Harman found none with surgical tuberculosis.

The figures given for positive von Pirquet reactions are high, but of doubtful value, since controls from a similar class of patient were not usually taken. It is significant that a proportion of negative results is obtained.

It is difficult to assess the value of experimental production of phlyctens with tuberculin. Gibson made an exhaustive series of experiments in 1918, and claimed to have produced phlyctens with tuberculin in tuberculous rabbits, but only at the height of a severe general conjunctival reaction. He would seem to have removed the affected eyes without waiting to watch the clinical progress.

There are some valid arguments against the tuberculosis theory. Although tuberculosis is said to be associated with phlyctens, phlyctens are not associated with tuberculosis. Dr. Hebert, the physician to the tuberculosis department at Saint Thomas's Hospital, could not, in 1926, recall having seen a case, and was convinced that it did not occur to any material extent amongst his patients; and Savin and Preston mention that, at Victoria Park Hospital, amongst the notes of 240 tuberculous children there was no record of phlyctenular disease.

Goldbach suggested that it is seen only in commencing tuberculous infection and not when the disease is fully established, but the history of some patients extends over many years, and therefore the suggestion seems to me inadequate.

Lastly, if tuberculosis is the cause, it is difficult to explain why babies are exempt, and why females are affected more often than males.

Eyre investigated 206 cases of tuberculosis of the conjunctiva and Lundsgaard 48 cases. They do not mention phlyctens as having occurred in any case.

Toxæmia from Causes Other than Tuberculosis.—Chronic infection of such tissues as the tonsils has sometimes been put forward as a cause. In my cases, removal of diseased tonsils and adenoids did not have any effect on the eye condition. Of 60 children with relapsing keratitis examined by Harman for transference to the County Ophthalmia School and referred back for removal of diseased tonsils

and adenoids, 45 did not need transfer after the operation, 12 did. The 12 are, I think, no less significant than the 45, for temporary improvement in institutions and intervals of relief are a feature of the disease.

Intestinal toxæmia has been cited as a cause. Constipation and indefinite abdominal discomfort, for which dietetic error seems to be the most likely explanation, certainly seem to be common.

Fifth Nerve Irritation.—Harman concluded that the disease is primarily constitutional, but that fifth nerve irritation from infected mouths, nasal catarrh, herpes and impetigo plays a part. That two factors, a constitutional and a local, are responsible does seem very probable, but in my cases the proportion of local infective lesions was not so large as to appear significant. My evidence, so far as it goes, suggests rather that strain from refractive error may be a local factor of importance.

Diet.—This disease can scarcely be explained on the grounds of local irritation alone, and, although the patients do not conform to any particular type, it is necessary to postulate some constitutional predisposition. Evidence is difficult to obtain, but it is very possible that this predisposition may be the consequence of dietetic error. The suggestion is based upon such fragmentary information as could be elicited from patients or their parents, and upon the favourable results sometimes obtained by restricting the sugar in the diet.

De Schweinitz, while thinking that the disease is tuberculous, mentions diet as a possible predisposing cause, and Duane, suggesting a metabolic disturbance, states that it would seem that regulation of the diet alone may do a great deal towards effecting a cure.

A dietetic theory would explain the association with poverty and the success of institutional treatment. Again, it would perhaps explain the absence of phlyctens in young babies, and, lastly, such excess of infective conditions as is found in phlyctenular patients may well be secondary to nutritional disorder.

The most likely dietetic error, especially in regard to the children of the poor, is either a want of balance with carbohydrate excess or a vitamin deficiency.

In correction with metabolism it is worth while remembering that rosacea keratitis bears many resemblances to phlyctenular keratitis.

Refraction.—Further investigation of the refraction in phlyctenular disease is required. The presence of refractive error sufficient to cause asthenopia would explain several clinical points in regard to the keratitis: (*a*) the chronicity; (*b*) the association with measles, for measles commonly unmasks errors of refraction; (*c*) the intolerance to light after the cornea has apparently healed; (*d*) the benefit derived from paralysing the accommodation.

De Schweinitz remarks:

> There is reason to believe that astigmatism may be very potent in the production of keratitis in children.

And Eyre, in his Hunterian lecture on tuberculosis of the conjunctiva, says, in passing:

> I see no valid reason for regarding phlyctenular conjunctivitis, *per se*, as a form of tuberculosis of the conjunctiva. I rather regard phlyctenules as an expression of enfeebled vitality and lowered cell resistance in an individual whose ocular system is habitually in a state of irritable tension.

Fresh Air and Sunlight.—Lack of fresh air and sunlight is generally assumed in England to play at least some part in the ætiology, an assumption based on the prevalence of the disease in smoke- and fog-laden cities, the unhygienic clothing of many of the children, and the frequently favourable response to general phototherapy. This assumption, however, is certainly not supported by the cases I have seen in Auckland, and it seems to me unlikely that it could have been a factor of importance before the industrial era.

I think it would be fair to say that in London the chief sufferers from over-clothing and from lack of air and light are babies, and these seem not to be liable to the disease.

Bacteriology.—Bacteriology and pathology have so far been unhelpful in the elucidation of the problem, and, while the appearances suggest an infection, the relapsing clinical course and the absence of contagion are against such a possibility. It is, however, conceivable that a virus infection is present and that the corneal lesions are trophic. It seems hardly likely that the herpes virus is responsible, and it may, I think, be said that what little we know of virus infections does not help us to explain phlyctenular ophthalmia.

CONCLUSION.

In approaching a problem such as the one under discussion it is necessary to consider the possibility of a combination of causes. It is usually unphilosophical to think of a single cause for a disease, and in the case of phlyctenular ophthalmia it is not unlikely that it is in this way that the solution is obscured.

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SALPINGITIS.[1]

By CHARLES M. GREENSLADE,
Dunedin.

I AM approaching a controversial subject from an unusual angle. My subject today is that particular variety of salpingitis which results from the ravages of that ancient, cosmopolitan, pertinacious and elusive organism, the gonococcus. It is no new disease. According to Luys, the oldest description of gonorrhœa dates back to the fifteenth century B.C., and runs as follows:

> Speak unto the children of Israel, and say unto them, When any man hath a running issue out of his flesh, *because of* his issue he *is* unclean.—*Leviticus*, XV, 2.

Ricord goes back further. He says:

> In the beginning God created the Heavens, the Earth, Man and the Venereal Diseases.

Although it is a disease of humans only, it is so cosmopolitan that no race, no country is free from its manifestations. Its pertinacity needs no special emphasis. Of its elusiveness let me point out that one or two negative results from examination, especially in mild or chronic cases, do not exclude its presence. Repeated examinations by smears and cultures, more particularly of urethral and cervical discharge, best taken after a period, are essential in doubtful cases. The Great War brought us to a realization of its devastating effects on the male. It is only since then that we have grown to appreciate that it is not one whit less dangerous to the female. A few years ago the medical curriculum hardly provided any instruction, theoretical or practical, in what must be regarded as one of the more common diseases, and this is one reason why the diagnosis of gonorrhœa in the female is so often missed.

During the last four years there have come under my notice seven patients who had acute gonorrhœal salpingitis, and whose appendices on removal were for all practical purposes normal, and were proved normal by pathological examination. As the pathologist usually manages to find some disease in the appendix of an adult, and, as in some doubtful cases there has been no pathological report, this does not represent, by any means, the total number of such cases that I have encountered during this period. The mistake is not an uncommon one, and it is not serious, provided the cause of the salpingitis is realized and treated. One of my reasons for speaking to a meeting of surgeons on this subject is

[1] Read at the annual meeting of the Royal Australasian College of Surgeons, Auckland, January, 1937.

that the surgeon is just as likely as the gynæcologist to be faced with the management of this condition, or at least to be responsible for the diagnosis and immediate treatment; and of all the common abdominal emergencies salpingitis is the one that is treated with the least skill. Further, to prove this contention, I have selected from my notes three different cases which had been treated, not by general practitioners, nor in our smaller hospitals, but by members of the staffs of metropolitan hospitals. Unfortunately, these are not isolated examples.

CASE I.—A farmer was referred to me on February 4, 1930, with acute gonorrhœa of ten days' duration—his first attack, an unwelcome souvenir of his recent marriage. His wife was with him, and she proved to have a gonococcal cervicitis and urethritis with a fairly profuse discharge. Here is her history. She was in hospital on May 20, 1927, and underwent appendicectomy for an acute attack of appendicitis. The pathological report reads: "Appendix dilated; no inflammatory changes now present." She was readmitted to hospital on July 30, 1929; her condition was diagnosed as acute salpingitis, and she was treated by rest and abdominal packs. On August 8 the cervical discharge showed "Gram-negative diplococci with the characteristics of gonococci". She remained in hospital without treatment to the cervix or urethra till September 7, when she was discharged to report to the gynæcological out-patient department for observation, but she was readmitted to hospital on September 18. On September 25 a double pyosalpinx was removed, also the left ovary. She was discharged on October 15, 1929. All that she knew when she was brought by her husband to me on February 4, 1930, was that she had had an operation for "peritonitis with abscess". She said she did not bother about the vaginal discharge, as a lot of women suffered from "whites". She said that she did not know that she had had gonorrhœa, nor that she was unable to have children. The husband was an uncomplaining fellow, bore his wife no great grudge, and so they both underwent treatment, with satisfactory results.

There is an old saying: "The woman always pays." The husband did in this instance.

CASE II.—A single woman of thirty years reported to the clinic with gonorrhœa and secondary syphilis on February 14, 1934, and left for another metropolitan hospital on March 24. She returned on December 4, 1935, with a suprapubic sinus discharging pus and a mass (the size of a tennis ball) above the uterus. Pressure from above and below caused pus to well up freely. She was wasted, weak and in very poor health. She said that she had been operated on six months previously, and that the sinus had persisted since the operation. She had had injections for the syphilis, and dressings for the sinus. Lipiodol was injected into the sinus. X ray examination showed the lipiodol running down into the pelvis and spreading laterally into two masses. At operation, on December 9, a sinus as thick as the little finger was found running down into the pelvis, attached in front to the bladder and uterus, behind to the sigmoid colon, and appeared to enter and run through a piece of small intestine. There were dense adhesions. The pouch of Douglas was obliterated. A left tubal abscess and a right tubo-ovarian abscess were removed with the sinus in one piece. A lateral anastomosis was necessary to restore the lumen of the gut. The appendix, which lay retroperitoneally behind the tubo-ovarian abscess, was removed, and numerous intestinal adhesions were divided. She was discharged from hospital on January 17, 1936, and attended the clinic till February 29, when she was free from infection; but, as she was leaving for another town, she received instructions to report for observation there.

CASE III.—A single woman, aged twenty-five years, was admitted to hospital with acute abdominal pain following a menstrual period which was worse than usual. Laparotomy disclosed acute bilateral suppurative salpingitis. A drain was inserted into the pelvis, and was removed on the fourth day. The pulse rate

remained in the vicinity of 100, and the temperature was intermittent, rising to between 37·8° and 38·3° C. (100° and 101° F.) each evening. On the twenty-seventh day the abdomen was again opened and both tubes were removed; the appendix was also removed. The pathologist reported "acute suppurative salpingitis", and stated that sections of the appendix showed no gross changes. The pelvis was drained both vaginally and abdominally, the vaginal drain being removed on the sixth day and the abdominal on the thirteenth day after the operation. Thirty-one days after the second operation—fifty-eight days in all—the patient was sent to us for treatment to the cervix and urethra, and this was the first cervical and urethral treatment she had during her illness.

This is a good example of the futility of draining the pelvis in acute suppurative salpingitis; this patient had a profuse and distressing vaginal discharge until she received lower genital treatment. Two months' active treatment cleared the infection, and observations after a further three periods showed that she remained free from the disease.

In limiting my remarks on salpingitis to those varieties resulting either primarily or secondarily from gonococcal infection, I am really not restricting my subject much, for Curtis[1] showed after an exhaustive analysis of nearly 300 infections that 70% were gonococcal, 10% were thought to have been primarily gonococcal, 5% were tuberculous (that is, in the absence of generalized tuberculous peritonitis), and 15% were due to other pus-producing organisms, notably streptococci. As a primary cause of salpingitis neither the colon bacilli nor the staphylococcus appeared to be of notable importance.

In the adult female the gonococcus may attack the urethra, the paraurethral ducts, Skene's tubules, Bartholin's duct and gland, and the cervix, and produce from any or all of them a purulent discharge. The urethra and the cervix are the commonest sites of infection. The vagina is rarely affected, unless the entrance is so narrow as to interfere with free drainage of the discharge. The disease, as in the male, is more a genital than a urinary infection; rarely does it pass the trigone of the bladder in either sex. The spread in the male is via the prostate, seminal vesicles and vasa to the epididymis; in the female, the narrow internal os, with its plug of mucus, forms a natural barrier to the ascent of the gonococcus, but once it passes this it travels upwards through the uterus without much resistance into the narrow tubes and so to the ovaries.

The tubes offer a strong natural resistance to the onslaught of the organism, and the readiness with which adhesions are formed between the fimbriated ends and the ovaries greatly tends to localize the disease to the pelvis; occasionally, however, there may result a moderate degree of peritonitis, and sometimes one encounters filmy adhesions even between the liver and the diaphragm. The direct result of tubo-ovarian infection, as with vaso-epididymal infection, is blockage of the genital duct. This makes gonorrhœa the greatest of all sterilizing diseases.

While the clinical appearances of gonorrhœa may be fairly typical in an early case, the diagnosis must always be confirmed by the finding of the Gram-negative bean-shaped intracellular diplococci, either in direct smear or by culture, and in mild infections this may entail

repeated examinations of the discharge. The best times to secure positive findings are either just before or just after a period. The patient should be instructed not to use a douche for twenty-four hours beforehand, and to retain her urine for at least two hours prior to being examined. In such circumstances, if the urethra is infected, the discharge can be massaged forward from the vaginal aspect (see Figures I and II), and the organisms fairly easily demonstrated. Vaginal smears are useless owing to the multiplicity of other organisms present. A smear from the cervix needs to be taken from within the lumen after wiping off the discharge from the surface of the external os. Pus may be milked down Bartholin's duct (see Figure V), or squeezed up from Skene's tubules (see Figure IV); but it is to be noted that infections of either of these sites are not necessarily gonococcal. Smears from the paraurethral ducts are not usually satisfactory owing to contamination with the vaginal flora. Provocative measures, such as the passage of instruments, glycerine packs and vaccine injections, are sometimes of assistance. Localized reactions from intradermal vaccines cannot as yet be classed as reliable evidence, and the complement fixation test is available only to the few who are in contact with a laboratory specializing in this work.

I cannot too strongly stress the fact that the only satisfactory position for the examination of the genitals is the lithotomy position. A good light is essential. A consulting room which is not equipped with a lithotomy couch, so that the patient can get into position easily, and remain there comfortably, is insufficiently furnished, and is the cause of many patients being incompletely examined, and of some conditions, no doubt, being incorrectly diagnosed. It is almost impossible to examine or to carry out local treatment to the urethra and the cervix with the patient in any position other than the lithotomy position. The late Frank Kidd stated that the lateral or Sims's position was the curse of English gynæcology. The days of prudery have passed. Nowadays women, tactfully handled, do not object to the lithotomy position.

Any factor, such as heavy work, undue fatigue, alcohol, sexual excitement, injudicious vaccine therapy or debilitating illness, which lowers the resistance of the body to the gonococcus, favours the development of salpingitis, just as in the male it often results in prostatitis, vesiculitis and epididymitis. The normal uterus draining well is a good safeguard against the extension of the disease, but when it has fallen backwards into the pelvis and is, as a consequence, draining badly and congested, a tubal infection will persist. The gonococcus spreads along mucosal surfaces and in the submucosa, rarely by lymphatics, sometimes by the blood stream. The narrow internal os, with its plug of mucus, if it is not interfered with imposes a very definite bar to the ascent of the gonococcus; however, instrumental dilatation of the os or forcible douching will break down this natural resistance and allow the organism to gain access to the uterus and so onwards into the tubes. Menstruation, miscarriages and childbirth, by opening the os providing a large raw uterine wound surface, are specially dangerous

events for a woman with gonorrhœa. Anything which stirs up the lower genital infection, most particularly coitus with its consequent pelvic congestion, is productive of salpingitis. Coitus indulged in about the time of the period simply invites trouble. Salpingitis is a disease of exacerbations. The gonococcus, as Curtis has proved, dies out usually within two weeks of producing the acute attack. Repeated insults from the factors mentioned above wear down resistance to such a point that the tube succumbs to the unequal struggle. An uncured partner is a continual source of danger. It seems ridiculous to have to mention it, but, time and again, I have come across cases of chronic gonorrhœa due to the fact that only one partner was receiving treatment. As with males, some females seem to have extraordinarily little resistance to the gonococcus.

CASE IV.—The patient's husband reported to me on April 26, 1936, with an acute Neisserian urethritis, the result of an extramarital intercourse four days previously. He first noticed his discharge shortly after coitus with his wife, twenty-four hours prior to consulting me. I examined his wife next day and found no macroscopic or microscopic evidence of infection. Two days later I found gonococcal pus in the urethra and in Skene's tubules. There was a muco-purulent cervical discharge, but I was unable to find any gonococci in it at this stage. I left for a few days' vacation and returned to find that a colleague had sent the patient to a nursing home with an acute salpingitis. This showed no signs of subsiding with expectant treatment, and, as she was anxious to get out of the hospital, I opened her abdomen on May 19, and removed a large left tubo-ovarian and broad ligament abscess adherent to the pelvic colon, with tube and ovary in one piece, and a greatly swollen right pus tube which had fallen back into the pelvis. On account of oozing, a drain was left in the pelvis. She left the nursing home twenty days later, and from June 23 was free from infection. She was examined after periods in July, August and September, and found free from infection clinically and bacteriologically.

While salpingitis may arise during the course of routine treatment to the urethra and cervix, and be cured without becoming anything more than a mild complication, when it produces great disturbances of health, the patient generally falls into the hands of the gynæcologist, and in the presence of more acute manifestations not infrequently comes under the care of the general surgeon. While there may be no lack of appreciation of the fact that salpingitis is usually a complication of gonorrhœa (although a recent English textbook barely mentions it), it does not seem to be recognized, as it should be, that if the tubes are the seat of this disease, the cervix must be infected and definitely in need of treatment; the urethra and other genital foci will probably be infected and in need of treatment also, even though the patient may be quite unaware that she has an infection.

CLASSIFICATION OF GONORRHŒAL SALPINGITIS.

To crystallize our thoughts it is convenient to classify gonorrhœal salpingitis as:

1. Catarrhal: (*a*) acute, (*b*) chronic.
2. Suppurative: (*a*) acute, (*b*) chronic.

Acute catarrhal salpingitis is an inflammation of the tubal mucosa with the formation of a clear serous discharge. Repeated attacks may cause sealing of the fimbriæ and the formation of a hydrosalpinx. Clinically, the main feature is pain in the lower part of the abdomen. The pain is situated just above Poupart's ligament and suprapubically. It may vary in intensity from a mild ache to one of such severity that the patient is regarded as presenting an abdominal emergency. Although the pain may be severe, yet there may be no pus formation. The temperature does not rise above 37·8° C. (100° F.). The pulse rate is not much affected. The attack commonly follows a menstrual period. Tenderness is found above Poupart's ligament and in the suprapubic region. Bimanually the uterus is tender on manipulation and tenderness is present in the lateral and posterior fornices. In chronic catarrhal salpingitis from repeated mild attacks the tubes become thickened, sclerosed and nodular. The ovary becomes inflamed, and pelvic adhesions are common.

Clinically, there is hypogastric and iliac pain associated with low backache, made worse by effort, especially heavy lifting. Increased premenstrual pain and menorrhagia are common complaints, although in some instances, apart from debility and loss of weight, little discomfort has been noticed by the patient. There is usually some vaginal discharge. Coitus may be painful.

In acute suppurative salpingitis the inflammatory reaction in the tubes is much more pronounced than in the catarrhal variety. The tubal mucosa rapidly becomes swollen, exuding at first serous, later purulent, fluid, which, owing to its proximity, involves the ovary, and which may escape into the peritoneal cavity, giving rise to pelvic peritonitis. The tube itself becomes greatly swollen and dark red in colour, is covered with lymph and rapidly becomes adherent to ovary, intestines and other pelvic viscera. The cellular tissues of the pelvis become congested and œdematous, sometimes thrombosis of the veins of the broad ligament occurring. Clinically, the symptoms are those of the catarrhal variety, only much more intense. The pain is more severe, the temperature rising higher, to 38·9° to 39·4° C. (102° to 103° F.), and tending to become intermittent, with morning drop and evening rise. The general appearance is that of peritonitis, with rapid pulse, dry furred tongue and tenderness and rigidity in the lower part of the abdomen. Vomiting is a bad sign, and usually means an extension of the peritonitis to the abdominal cavity. Uterine hæmorrhage is common; vaginal discharge is copious. Bimanually the cervix may be soft, the uterus enlarged slightly, tender on movement, and frequently retroverted. Tenderness is acute in lateral and posterior fornices.

In chronic suppurative salpingitis the fimbriæ of the tube, which in the acute stage tended to curl in and become adherent, have now closed off the abdominal ostium to form a pyosalpinx. The close proximity of ovary renders it liable to become infected by direct extension of organisms. The fimbriæ become adherent to the ovary, the result being a tubo-ovarian abscess. If the pus gravitates to the pelvis

a localized pelvic abscess may form. In most cases the heavy distended tubes fall back into the pelvis, dragging the engorged uterus with them, a common finding at operation being a tubo-ovarian abscess with a swollen retroverted uterus. A large tubo-ovarian abscess may fill the pelvis and so keep the uterus from falling back.

Clinically, the patient looks washed out, anæmic, has lost weight, is depressed and easily tires. Tenderness is found low down in the abdomen, and resistance to pressure is noted. Bimanually the lateral and posterior fornices are tender. The cervix may point forward or to one or other side. It may be difficult to distinguish the shape of the uterus owing to the pelvic exudate. It is often retroverted. It may be possible to make out a thick swollen tube or a tubo-ovarian abscess as a tender mass lying to one side of and behind the uterus.

DIAGNOSIS.

The salient points in the diagnosis of acute salpingitis are as follow.

1. On careful inquiry it will be found that there has been some disturbance in the last menstrual period or, in more chronic cases, in several. Usually there has been rather more menstrual flow; the flow may have lasted a little longer; there has been more pain than usual. In acute suppurative salpingitis there may be so much bleeding as to confuse the diagnosis with that of rupture of an ectopic pregnancy; but in this condition there is a history of one or two missed periods, a more abrupt onset, greater shock, low temperature, and sometimes the passage of a piece of membrane.

2. Dysuria is a common finding. The attack may have been transient, and is due to the initial stages of the urethral infection. Parous women with wide gaping vaginæ may miss urethral infection. Recurrent attacks of urinary discomfort are suggestive either of salpingitis or pyelitis. Dysuria is not common with appendicitis.

3. The pain is localized well down, lower than in appendicitis, and may be on both sides. Epigastric pain is uncommon. The sequence of epigastric pain, followed by vomiting, and later the localization of the pain in the right iliac fossa, with tenderness and rigidity over McBurney's point, so common in acute appendicitis, is not found.

4. The history of a vaginal discharge, or its discovery, demands an investigation of the lower genital tract. Neglect of this precaution is the commonest cause of inflicting on the patient an unnecessary operation, with its consequent economic loss and often harmful results to her pelvic anatomy.

5. Abdominal rigidity is low down, just above Poupart's ligament and in the suprapubic region. It is not so absolute as in appendicitis.

6. The bimanual examination reveals alteration in the position of the cervix, tenderness in the fornices, changes in the uterus, softening and enlargement of the acute stages; sometimes the distinct fluctuation of an abscess.

Here let me digress a moment to interpose a warning. During the last few years gonorrhœa of the rectum in women has been proved to be more common and more harmful than was ever imagined. Observers vary as to the frequency of its association with genital gonorrhœa from 4% to 75%. Martin[2] found the incidence in his series from 1929 to 1934 to vary between 25% and 42%. "Never insult the vagina by examining the rectum first" was an old teaching tag; however, owing to the very definite risk of adding a rectal to a genital infection, the glove should be changed if both orifices are to be examined.

Of other causes of salpingitis the most common is the streptococcal, which usually follows a septic delivery, or abortion, or an attempt at one. The infection extends through the uterine wall into the lymphatics and cellular tissues of the pelvis, in contradistinction to gonorrhœa, in which the spread is mucosal, and results in a pelvic cellulitis. The tubes are infected mainly on their peritoneal surfaces, the mucosa often being little affected. A sudden onset following delivery, abortion or instrumental attempts at abortion, with generalized pain of a greater severity than that of a gonococcal salpingitis, a faster and feebler pulse with higher temperature, and more tendency to the development of general peritonitis, are suggestive of a streptococcal infection. A foul vaginal discharge may be present; the patient looks much more ill and toxic than the gonococcal patient, and probably shows none of the lower genital lesions of gonorrhœa. No pains should be spared to arrive at an exact diagnosis, especially in acute salpingitis, for with few exceptions the immediate treatment is non-operative. The commonest conditions confusing the issue—acute appendicitis, ectopic pregnancy, torsion of an ovarian cyst—require urgent surgical intervention. In acute salpingitis operation causes in nearly every case an unnecessary economic loss, as well as an unnecessary interference with the pelvic anatomy, and has but one redeeming feature—that an appendix which possibly might become troublesome, as might any normal appendix, can be removed without much extra risk.

In those patients who are so ill and whose distress is such that it is difficult to get an accurate history, or to conduct a satisfactory examination, and when there are grave doubts as to whether the condition is an acute appendicitis going on to peritonitis, or an ectopic pregnancy with severe hæmorrhage, it is better to open the abdomen than to risk losing the patient owing to a mistaken diagnosis.

If one is treating a patient for gonorrhœa there is not much difficulty about the diagnosis; but when the surgeon is confronted with a young woman of whose habits he is unaware, and when he has to examine her in front of the mother or sister, the question of salpingitis arising, the best procedure is to admit her to hospital for observation, put her on an hourly chart, and at the first convenient opportunity get a full history, and, most important of all, make a thorough examination.

TREATMENT.

The immediate treatment of acute catarrhal salpingitis and of the majority of cases of acute suppurative salpingitis is non-operative. In mild cases the patient should rest as much as possible while continuing with topical applications to the lower genital foci. Rest is imperative during the menstrual periods. Plain food, plenty of fluid, no alcohol

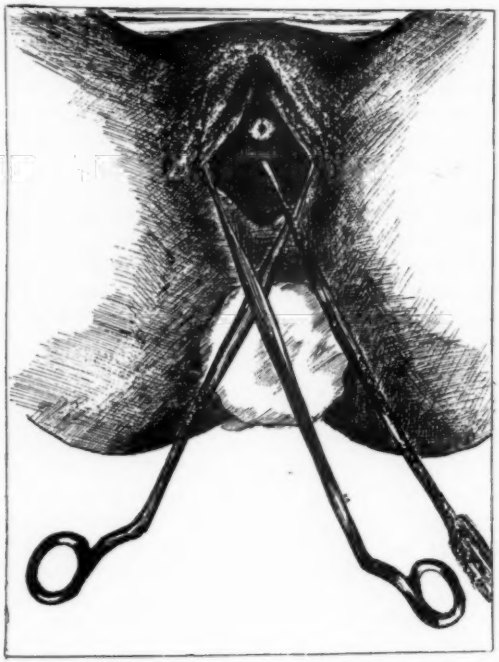

FIGURE I. Exposure of the urethra. The patient is in the lithotomy position, the labia being separated with sponge-holding forceps. Any vaginal discharge about the meatus is removed by a sterile cotton wool covered probe. The second pair of sponge forceps has been introduced into the vagina. By pressing upwards and forwards on the urethra any discharge is milked towards the meatus. The anus is covered with a pad of cotton wool. No other protective cloths are necessary.

and no sexual excitement are essential. It is remarkable the numbers of gonorrhœics, both male and female, who will not abstain from coitus, let alone sexual excitement. During 1935 eight women attending out of 95 became pregnant during the course of treatment; eight were pregnant before commencing treatment. As a fair number probably took precautions to avoid infecting their partners, and as some would not become pregnant owing to the contraceptive properties of the

applications used and others on account of the amount of cervical discharge present, I am of opinion that about one in four would be a conservative estimate of those who will not refrain from intercourse. Spermatozoa are unexpectedly found sometimes in cervical secretions. Many males, likewise, with relapses during treatment, admit alcoholic indulgence and amorous adventures.

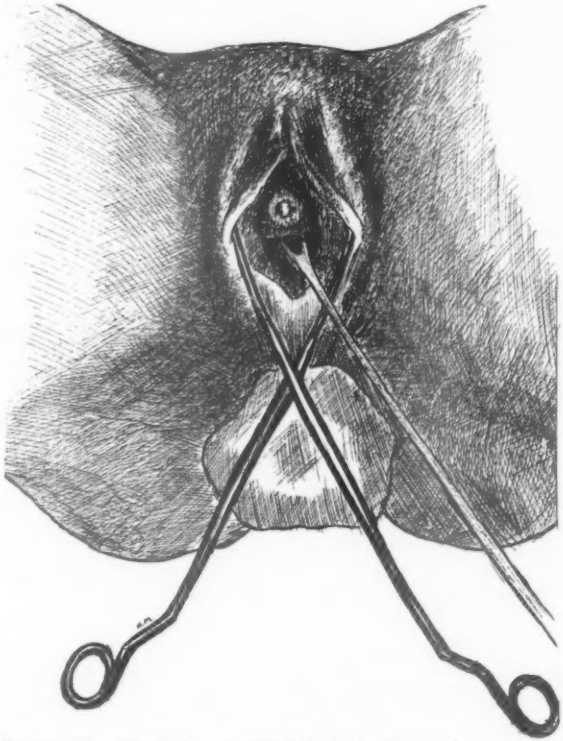

FIGURE II. Showing pus appearing at the meatus as the forceps is brought forward.

CASE V.—A divorcee, under treatment in 1935 for gonorrhœa, during my absence developed salpingitis. She was operated on by a colleague, who did not carry on the local treatment below, with the result that on resuming relations after the operation, her lover developed his first attack of gonorrhœa. They both came to me, and were curious to know why he had not become infected before.

Evidently the treatment she was having previous to operation was sufficient to make her a safe companion. Irresponsible individuals, especially of the hospital class, whose treatment entails no financial sacrifice, cannot be persuaded to refrain from indulging their passions.

Vaginal douches of weak antiseptics given as hot as can be comfortably borne may give relief; but, if they cause pain, they should be absolutely forbidden, as it is evidence that the douche is not being used properly. I have found daily intravenous injections of 18 centigrammes of "Sulfarsenol" of assistance. "Aolan", ten cubic centimetres, given intramuscularly, vaccines and calcium preparations have their advocates.

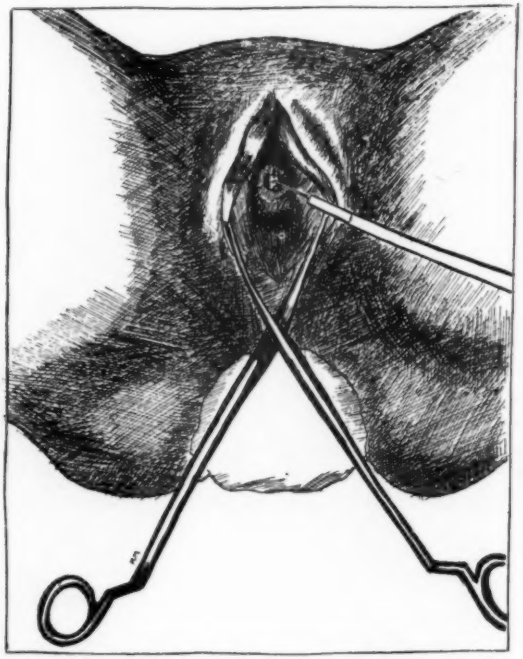

FIGURE III. The platinum loop is in position, securing the uncontaminated urethral discharge, which will be transferred to a slide for examination by Gram's method. A culture may be made on a blood agar plate.

Mild salpingitis subsides rapidly with this treatment faithfully carried out, leaving practically no macroscopic tubal changes, except perhaps a few filmy adhesions. I have been able to prove this in four cases in which I have treated patients for gonorrhœa complicated by catarrhal salpingitis, and have subsequently operated on them for other abdominal conditions.

Where the pain is severe, absolute rest in bed in the Fowler position is essential. Ice packs to the lower part of the abdomen in the early stages give great relief; hot packs are useful later. When there is no doubt about the diagnosis, it is well to relieve the pain with morphine

and atropine. The bowels should be kept open, if necessary by enemata, care being taken not to infect the rectum by carelessness with the nozzle. Such simple measures as these, faithfully carried out, will cause practically every case of acute salpingitis, catarrhal or suppurative, to become quiescent within from three days to three weeks.

Of special methods, diathermy is useful. It may be given in the mild

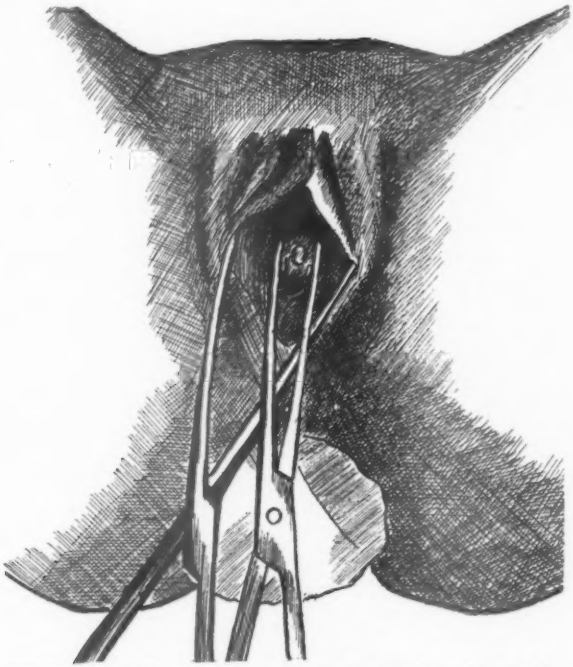

FIGURE IV. The forceps are shown compressing the urethra laterally. Pus may be expressed from Skene's tubules and the paraurethral ducts by this manœuvre.

cases with one electrode inserted into the cervix, the temperature of this electrode being raised as nearly to 45·5° C. (114° F.) as can be comfortably tolerated, the other electrode, a small plate, being placed over the painful area on the abdominal wall. It should be given for ten minutes daily until the pain subsides. A larger vaginal electrode may be also used instead of the cervical electrode. Bierman and Horowitz[3] report that the combination of systemic temperature elevation with simultaneous additional pelvic heating—photothermy and diathermy—has given excellent results in salpingitis. They report relief of salpingitis and in some cases cures of gonorrhœa with as few as three treatments. The systemic temperature is kept between 40·5° and 41·1° C. (105° and 106° F.) for one and a half to one and three-quarter hours.

A method in vogue at the Mayo Clinic is Elliott's method, in which a soft rubber vaginal applicator distended by circulating water at a temperature of 48·7° to 54·4° C. (120° to 130° F.) within it, is inserted and left in place for increasing periods, starting with half an hour daily. In those cases when the uterus is in good position, with no obstruction to free drainage, intrauterine injections of glycerine are effective.

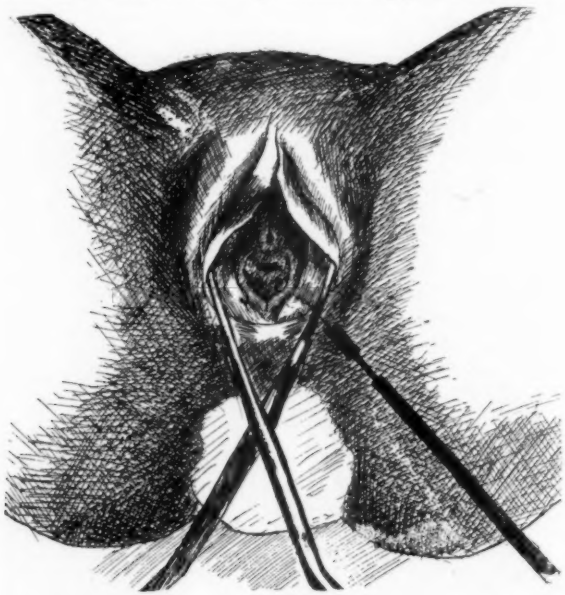

FIGURE V. Exposure of the orifices of Bartholin's ducts. Pressure from above downwards and inwards with the forceps will express any discharge from the ducts. The platinum loop is shown approaching the left duct orifice. This position of the forceps permits the easy introduction of the vaginal speculum, and avoids any contamination from the skin.

All patients with salpingitis should have treatment to the lower genital tract carried out as far as possible, even when they are acutely ill. For this a specially trained sister is necessary, and she requires a nurse to hold the patient in position for the few moments that are required to carry out the treatment. In Dunedin Hospital, with 310 beds, one sister is insufficient to carry out this work. How many hospitals in New Zealand have such a sister specially trained on their staffs?

Operative treatment in acute suppurative salpingitis is limited to those rare cases in which there is a spreading peritonitis; and I have yet to see a case which has been benefited by immediate operation, because if the patient receives appropriate treatment the disease will not spread, but localize itself to the pelvis.

One is forced to operate in acute cases in which there is doubt as to the exact diagnosis, and especially when appendicitis with spreading peritonitis is suspected or torsion of an ovarian cyst or distended tube. Such a patient, late at night, too ill to give a coherent history and too distressed to be examined thoroughly, is better explored. The incision should be a right lower paramedian incision so that both tubes

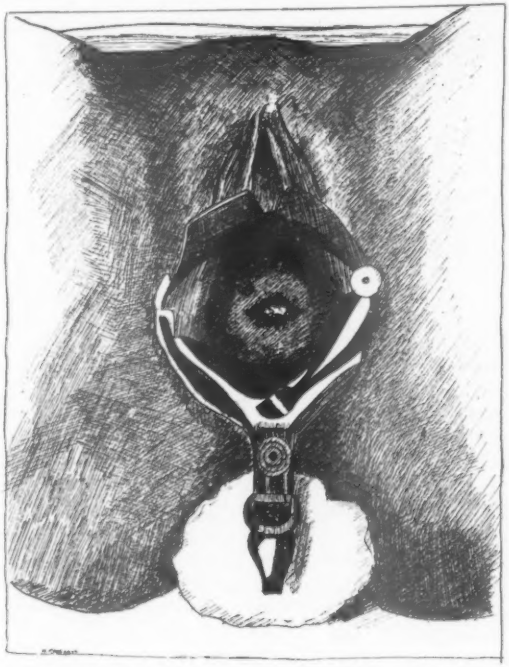

FIGURE VI. The Cusco speculum has been introduced, exposing the cervix. The discharge is swabbed from the external os, as it is usually contaminated with the numerous and varied organisms present in vaginal secretion. A platinum loop is passed into the cervical canal to collect discharge for microscopic examination.

can be seen. A right-sided grid-iron incision is unsatisfactory, as it does not permit adequate exposure of the left tube. Unless the tubes are grossly diseased, they should be left. The aim of gynæcological surgery is conservatism. I should except the case of the known prostitute; her tubes might as well come out while opportunity offers, as once she gets salpingitis, it will not be cured while they remain. Also when one tube is absolutely normal and the other has well marked disease, the diseased tube should be removed. The appendix should be removed. There is no need for drainage of the pelvis, if the tubes are left. If one tube or both should be removed, the only indication for

drainage is the oozing of blood from the friable cut edges. Pelvic drains invite the formation of pus. If the wound has been contaminated, it is wise to put a piece of rubber dam through the lower end as far down as the peritoneum, but no further.

Drainage of the pelvis is commonly advised in salpingitis, but is irrational. It is much better to drain the region between the fimbriated

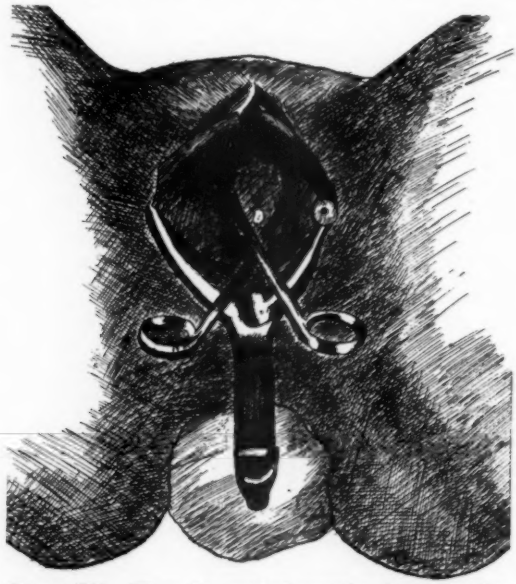

FIGURE VII. When the cervical secretion is scanty, the cervix is compressed between the blades of the sponge forceps. This expresses secretion from the cervical glands much in the same way as massage does from the prostate gland.

end of the tube and the ovary where there is a tendency for pus to accumulate to form a tubo-ovarian abscess. Salpingostomy is advocated by some, but I think the patient has a much better chance of getting a functioning tube the less its anatomy is disturbed in the presence of acute inflammation.

A more rational procedure, which I have not seen advocated, is to suspend a retroverted uterus, so that it can drain out its dammed-up pus. The heavy soggy uterus is by no means suitable for this procedure, but if it is left retroverted the chances of the salpingitis and endometritis clearing up are not good. Retroversion is one of the commoner causes of chronic gonorrhœa in the female. Free drainage through the natural passages is necessary in salpingitis. This is why the pus needs to be removed from the vagina and the cervix frequently during the course of suppurative salpingitis. This is why glycerine is such a useful agent;

being hygroscopic, it stimulates drainage. A patient who is not having treatment to the cervix does not get a fair chance. In fairness to the advocates of immediate operation in suppurative salpingitis, it must be admitted that after three or four weeks of conservative treatment one has to operate in a varying number of cases. Among these a certain number of patients are going to lose both tubes, and their convalescence is going to last just about as long as those who have their tubes removed at operation in the acute stage, so that from an economic viewpoint they have spent three or four weeks to no good purpose in bed. However, in the acute stage, lacking pre-operative preparation, with fever, rapid pulse, tissue dehydration, toxæmia and sometimes incomplete investigation, a patient is not such a good risk as one carefully prepared for a planned operation.

It is argued, too, that immediate operation permits the removal of one diseased tube, and by so doing lessens the risk to the healthy one. In my opinion it is the rest in bed and consequent segregation from the partner entailed by operation which give the healthy tube its chance of remaining healthy. With expectant treatment above and topical treatment below, such patients have a good chance of having two healthy tubes.

Chronic mild salpingitis with thickening and fibrosis of the tubes can often be greatly relieved, if the uterus is in good position, by intrauterine injections of glycerine or by pelvic diathermy; but when gross anatomical deformity or continuous pain is present, operation offers the only relief. In some of these cases division of adhesions, salpingostomy and replacement of the uterus may result in a restoration of fertility.

Chronic suppurative salpingitis with its complications—pelvic adhesions, pyosalpinx, tubo-ovarian abscess, cystic degeneration of the ovary, retroversion of the uterus, endometritis and pelvic abscess—practically always requires operative treatment for satisfactory results. In the presence of gross pelvic pathological change it is a mistake to spend much time on topical applications to the cervix, as Case VI illustrates.

CASE VI.—A well-known prostitute who had had two previous attacks of gonorrhœa cleared up at the clinic some years ago, was sent to the hospital by a general practitioner after three months' treatment to the cervix. During this period most of her time was spent in bed owing to the severity of the abdominal pain and backache. Bimanual examination disclosed a firm, tender mass filling the pelvis. At operation on November 18, 1935, I found double tubo-ovarian abscesses and an enlarged uterus, all bound together in one mass with adhesions to the pelvic colon, rectum, small intestines and appendix. The whole of the uterus, tubes, ovaries and appendix were removed. The patient was discharged twenty-five days later with instructions to attend the out-patient clinic, and when last seen on January 29, 1936, she was still free from infection.

The usual operative procedure for chronic suppurative salpingitis is as follows. If chronic cervicitis is present and the uterus is to be retained, the patient is placed in the lithotomy position and the cervix is coned out with the endotherm knife or with the Kimble-Jaros

electrode. A change is now made to the Trendelenburg position. The abdomen is opened through a subumbilical paramedian incision. Adhesions between omentum, small intestine and colon are divided to expose the inflammatory mass. The pyosalpinx, including the uterine portion, or the tubo-ovarian abscess, or the degenerate ovary is removed in one piece. The uterus is replaced by Gilliam's method, unless the round ligaments are too weak, when it is held forward by silk sutures. If an ovary is being left, it is advantageous to retain the uterus to avoid undue disturbance of the ovarian blood supply. Ovaries left without adequate blood supply undergo cystic degeneration. The appendix is removed, and the abdomen is closed without drainage if there is no oozing. If the uterus is removed, there is no advantage in leaving the cervix. If the cervix is retained, it should be cleared up later. Vaginitis sometimes gives trouble after total hysterectomy. It requires treatment, as does any remaining urethritis, Bartholinitis or Skenitis.

SUMMARY.

I hope I have been able to convince you:

1. Of the close relationship of salpingitis and gonorrhœa.
2. Of the value of non-operative measures in acute salpingitis.
3. Of the scope of the operation in chronic salpingitis.
4. Of the necessity of clearing up the lower genital and urethral infection while dealing with the salpingitis.
5. Of the importance of alterations in the menstrual flow, and of examination of the lower genital tract in the diagnosis of acute abdominal emergencies simulating salpingitis.
6. Of the need of cooperation between gynæcologist, surgeon and venereal specialist in the management of salpingitis.

In conclusion, the second object for which this College was established reads: "To promote the practice of surgery under proper conditions by securing the improvement of hospitals and hospital methods." From my remarks you will have gathered, I think, that the surgery of gonorrhœa in the female is not up to the standard of surgery in general. I believe that the establishment in each of the public hospitals of this country, and particularly in those outside the four main centres, of a department for the treatment of gonorrhœa and its complications, under the charge of a surgeon or gynæcologist, and provided with a specially trained nursing staff, would be a great advance in the direction of increasing their efficiency.

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ANÆSTHETIC FIRES AND EXPLOSIONS.

By Geoffrey Kaye,
Melbourne.

Reports of fires and explosions in operating theatres are not uncommon in medical literature. The main features of their causes and prevention should be known by surgeons no less than by anæsthetists, and it is for this reason that the present paper was compiled at the wish of the Executive Committee of the Royal Australasian College of Surgeons. At the same time, no attempt will be made to consider the applied physics and chemistry of inflammable anæsthetic agents; the discussion will be limited to purely clinical considerations likely to influence the daily work of the average surgical team. The subject matter will be discussed under the following headings: (i) the inflammability of anæsthetics, (ii) fires due to "external ignition" of anæsthetics, (iii) fires due to "static electricity", (iv) some observations upon theatre design.

The Inflammability of Anæsthetics.

It should be clearly understood, at the outset, that there are but two non-inflammable anæsthetics, namely, chloroform[1] and nitrous oxide. The latter has, however, the power of supporting combustion of any inflammable substance (such as alcohol or ether) which may happen to be present and to become ignited. Further, nitrous oxide is almost invariably administered in conjunction with oxygen, an ideal supporter of combustion. It follows, then, that whilst nitrous oxide and oxygen mixtures are themselves quite uninflammable and may be used with perfect safety for such procedures as electrical cauterization of tissue, the addition of ether renders them not merely inflammable, but highly explosive.

All the other anæsthetics are inflammable in greater or less degree, and in certain conditions (as when mixed with oxygen) may be highly explosive. Ether vapourized with air is inflammable; vapourized with oxygen, it is extremely explosive. Ethyl chloride is highly inflammable in air. The gaseous anæsthetics (ethylene, acetylene and cyclopropane) are all inflammable in air. They are, however, commonly administered mixed with oxygen, in presence of which they become highly explosive. Indeed, if the percentage of oxygen greatly exceeds that of the anæsthetic gas, the resultant mixture is capable of explosion so violent as to amount to detonation. A nitrous oxide and oxygen mixture, as mentioned above, is safe; mixtures of nitrous oxide, oxygen and ether are, however, as explosive as ethylene.

[1] Chloroform-ether mixtures are inflammable in proportion to the amount of contained ether.

FIRES DUE TO "EXTERNAL IGNITION" OF ANÆSTHETICS.

Perusal of the literature of anæsthesia reveals a long list of accidents due to ignition of inflammable anæsthetics from a variety of causes external to the actual anæsthetic apparatus employed. One may say, indeed, that the great majority of such accidents (particularly in this country and in England, as opposed to America) have been due to the "external" ignition of the type above-mentioned. It is instructive to consider the factors which, at different times, have led to fires or explosions. They may be listed somewhat as follow:

1. Open fires, naked lights, found mainly in country practice.
2. Gas sterilizers, especially if housed in the operating theatre proper; dental unit gas jets.
3. Cauteries, either of the "actual" type or the modern electro-coagulation type.
4. Electrical radiators.
5. Electrical motors, whether used to drive suction pumps or the air pumps of anæsthetic apparatus. Motors for this purpose should be of the totally enclosed type;[1] a very hot spark may be generated at the brushes, and, if not effectually shielded, may readily ignite inflammable anæsthetics.
6. Defective electrical switches, resulting in sparking, either on wall fitting, theatre lights, endoscopic instruments or similar electro-medical appliances.
7. Diathermy apparatus, which may produce an extremely hot spark, either across the spark gaps (which are usually effectually shielded) or at the point of contact of the electrodes with the patient.
8. The X ray apparatus, whether portable or stationary, which is capable of discharging very large sparks of extremely high voltage.

It is now opportune to consider certain individual anæsthetics in terms of the sources of external ignition which have been described.

Ether vapour is heavy and falls to the floor, along which it may run for a considerable distance to reach an open fire, electrical radiator or similar source of ignition. Ether should therefore be avoided when any such danger exists. It should likewise be avoided when the actual cautery or diathermy apparatus is to be applied to the patient's head, neck, mouth or pleural cavity; numerous fatalities have resulted from neglect of this obvious precaution. In cases in which cauterization must be effected, it is frequently suggested that a change be made from ether to chloroform prior to the application of the cautery. Since ether is excreted through the patient's lungs, the procedure must inevitably be hazardous for some time after the giving of ether has been suspended. For how long a time it is more difficult to decide. Fires, fatal to the patients, have been recorded as having occurred "several" minutes after the change to chloroform, but the writer is frankly unaware of what constitutes a safe interval. If ether must be used,

[1] A certain gap is, in practice, required to admit air for cooling purposes, but this can be very small, and if desired may be made quite safe by shielding it with fine-mesh copper gauze.

the following technique should be adopted. Anæsthesia is maintained through a Magill's wide-bore catheter, the patient's pharynx being tightly packed to exclude any possibility of breathing around, rather than through, the catheter. The exhalation valve is so arranged as to discharge beneath the drapings as far as possible from the site of operation. Chloroform is substituted for ether five minutes before the application of the cautery. In this way, any ether vapour contained in the breath after a lapse of five minutes will have a reasonable prospect of leaving the exhalation valve so far away from the mouth as to escape ignition. It is doubtful, however, if even this plan provides an absolute safeguard, and nitrous oxide or chloroform might well be preferred for all such cautery cases. The use of the cautery in more remote areas, such as the anus, would seem to carry a theoretical rather than a practical risk, provided that whilst it is being heated the instrument be kept altogether away from the head end of the patient.

Ether, then, has proved relatively safe if removed from dangers of ignition of the grosser type. The keeping of a limited supply of ether in the theatre proper and the holding of the main stock in a safe ante-room would likewise seem to be reasonable. The recorded ether fires have seldom endangered the personnel of the operating theatre, although the consequences to the patient have at times been serious. In general, the fires have been rapidly brought under control by smothering under wet towels, starting at the patient's face. Graver results have followed the ignition of mixtures of ether vapour and oxygen, which ignite more readily than those of ether vapour and air, and which do so with explosive force. Serious injury has been caused to patients, anæsthetists and theatre personnel in this way. It would seem reasonable, therefore, to use ether-oxygen mixtures only where directly indicated and not (as is frequently the case in England) as a routine procedure.

The gaseous anæsthetics (ethylene, acetylene, cyclopropane and nitrous oxide with oxygen and ether) diffuse rapidly into the air of the operating theatre, so that, if the latter be well ventilated, explosive concentrations will be unlikely to be reached. The danger zone, then, will remain close to the mask, probably within a radius of a foot from it. Within that zone any source of ignition (such as a sparking electric light switch or endoscopic lamp) may excite an explosion likely to have the gravest consequences to everyone in the theatre.

Gaseous anæsthetics, then, with the single exception of nitrous oxide and oxygen, are absolutely contraindicated when the actual cautery or diathermy apparatus is to be employed. In the case of nitrous oxide a difficult position arises in the event of some measure of ether supplement being necessary to secure an adequate depth of anæsthesia. In such case the anæsthetic mixture will become explosive and will remain so for some minutes after the ether has been withdrawn. The onus, therefore, falls upon the anæsthetist to foresee the use of the cautery or diathermy apparatus and to discontinue the use of ether as long as possible beforehand. Indeed, when the diathermy apparatus is

being repeatedly used, as in cerebral surgery, the anæsthetist is well advised to disconnect one of its terminals so that it cannot be brought into play without his knowledge.

It is not generally realized by surgeons that the cylinders used for the storage of nitrous oxide, ethylene, oxygen and similar gases are filled to a pressure which in some cases approximates to half a ton per square inch of surface. Such a cylinder, if exposed to heat or violence, may explode with great force. Gas cylinders, therefore, should be handled gently and stored in a place removed from direct sunlight or heating appliances. A pipe line system, in which the cylinders are housed in a central storehouse and piped to the individual operating theatres under reduced pressures, is thus in the interests of safety.

In general, it is probable that the safety of gas anæsthesia will be materially increased by the growing use of carbon dioxide absorption methods of administration. Here the patient's respiration takes place in a closed circle within the apparatus, the anæsthetic mixture being confined to the latter and not entering the atmosphere of the theatre in appreciable amounts.

In conclusion, the prevention of fires due to external ignition of the anæsthetic mixture is purely a matter of care in detail, in which surgeon, anæsthetist and theatre staff must cooperate. The choice of anæsthetic must be appropriate, especially in operations involving the cautery or diathermy apparatus, and particularly in operations performed under X rays. The surgeon should warn the anæsthetist before making unexpected use of these agents. Both surgeon and anæsthetist should be personally responsible for the absence of loose connexions in their endoscopic instruments, laryngoscopes, electrical suction appliances and motor-driven anæsthetic machines. Endoscopic instruments are safer when operated from low voltage dry cell batteries rather than from transformers. The theatre staff, again, should be responsible for the safe storage of stocks of ether and gaseous anæsthetics, for the maintenance of pipe lines in good order without leakage and for the integrity of all wall switches, endoscopic instruments and electrical motors in its charge.

FIRES DUE TO "STATIC ELECTRICITY".

It is a familiar fact that the continued flow of gases over the rubber tubing of anæsthetic apparatus may result in the accumulation of "static" electricity. When this reaches a sufficient potential, it may be discharged as a spark to the nearest electrical conductor, and this spark may suffice to ignite the gas mixture delivered by the machine. Static charges may be also produced by friction, as by the rubbing of woollen clothing, walking in rubber soled shoes or pushing a rubber tired trolley over a floor. In either case, the static charge may become large enough to be discharged as a spark as soon as the charged body is brought into contact with a conductor.

The accumulation of static charges occurs most readily in cold, dry weather. In consequence, most cases of "static" have been reported from America, where such conditions rule in the winter time. The writer well remembers as a frequent phenomenon of the American winter the fact that, on walking down a carpeted hotel corridor in rubber soled shoes and inserting the key into the lock of his door, a visible and audible spark would leap from key to lock. It is not surprising to read, then, of many cases in America in which a gas machine, a patient on a trolley or even a theatre table when wheeled across the floor has resulted in a discharge of static electricity and an anæsthetic explosion. Static phenomena are apparently rarer in England, where winter temperatures are higher and where the humidity is greater than in America.

In Australia the climatic conditions for the accumulation of static charges do not generally exist. Accordingly, precautions against "static" (which are very elaborate in America, as befits a very real danger) may be to a large degree modified. In one Melbourne metropolitan hospital, where several thousand administrations of ethylene and nitrous oxide with oxygen and ether have taken place in the past ten years, no fire or explosion has resulted, although few precautions are taken. Nor is any case of fire or explosion from this cause, in any Australian clinic, known to the present writer. A few simple precautions are, however, reasonable and may be briefly indicated.

"Grounding" of Anæsthetic Apparatus.—Before use, gas anæsthesia apparatus should be "grounded" by running an intact copper wire from it to the nearest water pipe. A water pipe forms a good conductor to "earth" and will serve instead of the intricate metallic network incorporated in the floor, which is a feature of American operating theatres.

"Grounding" the Patient.—If the anæsthetist, before starting the flow of ethylene, will touch both his patient and the "grounded" gas machine, any static charge in either of them will be dissipated to "earth".

"Grounding" Theatre Equipment.—If rubber tired operating tables, trolleys *et cetera,* be fitted with a short chain trailing down to the floor, any static charge produced by moving them will be "earthed". The ordinary "granolithic" or concrete theatre floor is a sufficiently good conductor for this purpose. Further, members of the theatre personnel will automatically "ground" themselves when they touch these "grounded" appliances.

Humidification of the Atmosphere.—In the dry American winter considerable pains are taken to secure a mean humidity of the theatre atmosphere of some 54%, static accumulation being less under such conditions. In most Australian cities the normal humidity is sufficient. In any event, steam sterilizers effectually humidify the air. In the most modern hospitals, however, there is a tendency to supply "conditioned" air, which is relatively dry, and to remove the sterilizers from the operating theatre proper. Under such conditions, humidification of the "conditioned" air may be desirable.

Humidification of the Anæsthetic Gases.—Without entering on technical matters more proper to an anæsthetic journal, it may be pointed out that gas apparatus is of two kinds, namely, dry flow and water flow-meter. Of the former type the McKesson and Austox D-M machines will be most familiar to Australasian surgeons. The second type is characterized by sight-feed water flow-meters, and is illustrated by the Safety Gas, Foregger and Austox-Foregger machines. In this second type, inflammable gases such as ethylene are humidified in passage over the water flow-meters and additional safety is claimed in consequence. In the dry flow machines it is usual to incorporate a copper meshwork inside the delivery tube as a precaution against static accumulation, and to wet the inside of this tube before use. Where the carbon dioxide absorption method of anæsthesia is practised, the moisture of the patient's exhalations is conserved, so that the whole apparatus rapidly becomes moistened internally, providing a further precaution against "static".

It would seem that, of anæsthetic explosions recorded overseas, "static" has undoubtedly accounted for some and external ignition for others. Under Australian climatic conditions external ignition would appear more to be feared. Further, the sudden and violent thunderstorms of the Australian summer provide a purely local hazard, and one which causes the writer more uneasiness than does "static". With the near approach of a thunderstorm, a change from ethylene to nitrous oxide (or, if that be not available, to ether) would seem reasonable.

SOME OBSERVATIONS ON THEATRE DESIGN.

From the standpoint of anæsthetic fires, the following points in theatre design are perhaps worthy of consideration:

1. Theatre should be well ventilated and floored with a conducting material.

2. If the sterilizers are to be in the theatre proper, they should be heated by steam or by wholly enclosed electrical elements.

3. The warming of the theatre should be by central heating or by steam radiators. If electrical radiators must be used (as in small private hospitals) they should be of the lamp type, rather than the cone element. In either event, they must be extinguished should their presence imply risk of fire.

4. If "conditioned" air is used, and if the sterilizers are not in the theatre proper, humidification may be necessary.

5. A central storeroom for anæsthetic gases, combined with a pipe line system, is preferable to the use of cylinders in the theatre.

6. Switches for electrical light and power should be of an approved non-sparking design, and be placed outside the theatre proper.

7. Theatre lamps should be desirably of the single contact, screw-in type, which is less liable to sparking than the two-contact, bayonet socket type more commonly encountered.

8. Provision for suction and compressed air should be made in the pipe line rather than by the use of motor driven suction and air pumps in the theatre itself. The maximum of safety and silence are thus obtained, the suction pump and air compressor (or cylinder of compressed air) being housed in a conveniently central spot. If individual motors are necessary, they should be of the enclosed type, to obviate danger of sparking at the brushes. Desirably, they and their rheostats should be housed in an ante-room and connected by rubber hoses to the suction bottle and ether apparatus in the theatre proper. This will again make for silence and safety.

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Surgical Technique.

TREATMENT OF ILEUS, WITH SPECIAL REFERENCE TO CONTINUOUS STOMACH ASPIRATION AND MASSIVE INTRAVENOUS INFUSION.[1]

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By ileus is meant the dilatation of the stomach and bowel due to paralysis or paresis of the peristaltic mechanism. The term "adynamic ileus", given by Mason,[(1)] is very descriptive of the condition. Although ileus most commonly occurs after abdominal operations, it may appear in any state of generalized toxæmia, such as pneumonia or kidney failure. The purpose of this paper is to discuss some aspects of the treatment of ileus, with special reference to its occurrence after the operation of appendicectomy.

The following case history illustrates the alarming chain of events which may occur in an untreated case of post-operative ileus.

A.B., a male, aged twenty-five years, was operated upon for acute appendicitis of eighteen hours' duration. A right McBurney's incision was made, and an appendix showing catarrhal inflammation was removed without difficulty. There was no evidence of peritonitis and no other abnormality was seen. A preliminary injection of 0·01 gramme (one-sixth of a grain) of morphine and 0·54 milligramme (one one-hundred-and-twentieth of a grain) of atropine was given half an hour prior to the operation. Anæsthesia was induced by ethyl chloride and was maintained by ether given by the open method. A post-operative injection of 0·01 gramme (one-sixth of a grain) of morphine was given on the patient's return from the theatre. During the first twenty-four hours he vomited on five occasions; but this provided no occasion for any alarm, as he was very nauseated by the anæsthetic. On the second day after operation he vomited only once, and appeared to be progressing favourably, although his abdomen was slightly full. On the third day after operation he vomited twice, the vomitus consisting of undigested food mixed with normal gastric contents. This was attributed to a dose of salts which he had taken in the morning. The abdomen was more distended and there was no result to a soap and water enema. On the fourth day he vomited five times, about two pints in all, the vomitus being bile stained but not fæculent. The abdomen was still distended and there was no fæcal response to two enemata, but a small amount of flatus was passed *per rectum*. The patient was slowly becoming restless, anxious and very thirsty. His chief complaint was a sense of tightness in the abdomen and a constant desire to drink large quantities of water. The pulse rate was becoming more rapid and of poor volume, the systolic blood pressure being 100, and the diastolic blood pressure 70 millimetres of mercury. On the fifth day the vomiting was even more persistent, and quantities of about a half a pint were continually welling up into the patient's throat, causing him great anxiety. The vomitus was now becoming dark brown in colour, with the consistency of thick pea soup. The odour was strongly fæculent. His colour was slightly cyanotic, and his limbs were a little cold and showed

[1] Read at a meeting of the Royal Australasian College of Surgeons at Melbourne on July 30, 1937.

poor capillary return after pressure. His eyes and cheeks were becoming sunken. He was listless, anxious, excitable, but mentally he was lucid and taking a keen interest in his condition. During this day he vomited about ten pints of fluid. His abdomen was tense, especially in the epigastrium. The pulse was rapid and of poor volume. Enemata, bowel wash-outs and pituitrin were tried without result, and the patient developed a dry brown tongue, drowsiness, sunken eyes and hollow cheeks, cyanosis, air hunger, anuria, subnormal temperature, falling blood pressure and cold extremities. Twenty-four hours later he died.

Post mortem examination showed the peritoneum to be slightly injected, and there was only a small amount of clear free fluid in the abdominal cavity. The appendiceal stump was normal. The stomach, jejunum, ileum and colon were greatly distended, but no organic obstruction was present. The other organs, notably the kidneys and liver, showed cloudy swelling.

The cause of this state of paralysis of the bowel is not definitely known, and it is not proposed to discuss the various theories in this paper. The theory most commonly favoured is the formation in the body of a toxin, possibly of bacterial or hepatic origin, which paralyses the peristaltic mechanism of the bowel.

As a result of the paralysis, distension of the bowel with fluid and gas occurs, and vomiting sets in. The vomiting produces many important biochemical changes, and in order to appreciate these it is necessary to study some phases of the normal physiology of the alimentary canal and its associated secretions. Approximately 60% of the body consists of water which is situated in the cells, interstitial spaces and in the cavities of the body, especially the alimentary canal. Large volumes of fluid are continually being poured into the alimentary canal to mix with the food, and Rowntree[2] has estimated that in twenty-four hours as much as nine litres of water may be poured into the upper reaches of the alimentary canal by the saliva, gastric juice, bile, pancreatic juice and *succus entericus*. After passing to the ileum, colon and rectum, a similar amount of fluid is reabsorbed into the circulation. There is thus a continual circulation of fluid in and out of the alimentary canal, which has been aptly termed by Mason[1] the gastro-intestinal circulation (see Figure I). About three litres of fluid in the form of food and drink are added to this circulation each day and a similar volume of fluid is withdrawn from the circulation directly by means of the fæces and indirectly by way of the urine and sweat. In this way a balance is maintained which under normal conditions conserves an optimum amount of water in the body. When vomiting occurs from ileus large volumes of fluid are lost from the gastro-intestinal circulation, just as blood is lost from the systemic circulation during a severe hæmorrhage. The upper coils of the jejunum drain their contents back into the stomach, and these are vomited with the gastric and duodenal secretions. Moreover, not only is water lost during vomiting, but also food and salts, notably sodium chloride.

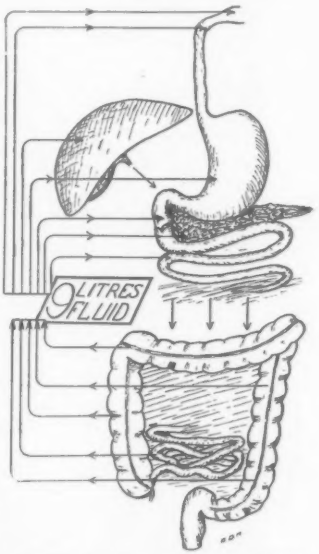

FIGURE I. The gastro-intestinal circulation. In a normal subject approximately nine litres (fifteen pints) of fluid pass through.

The patient thus suffers from dehydration, starvation and sodium chloride deficiency, and these losses are sufficient to explain many of the symptoms of ileus. Other biochemical changes have been recorded by Wangensteen,[3] Ochsner[4] and others. These have been summarized by Mason[1] as follows: (i) low blood

volume, (ii) fall in blood chloride, (iii) diminished output of urine, (iv) rise in blood urea, (v) alkalosis.

Treatment.

Prophylaxis and treatment by drugs *et cetera* will not be discussed in this paper, which will be confined to the description of measures which have usually proved adequate in drainage of the alimentary canal and replacement of the lost fluid.

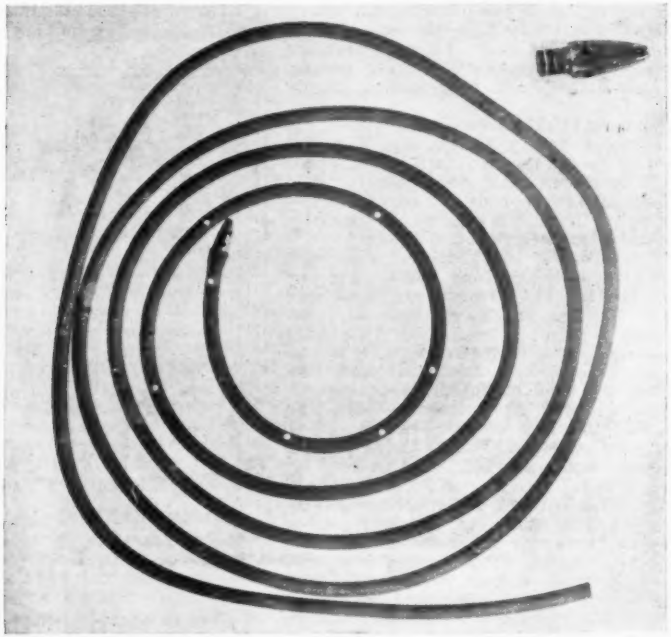

FIGURE II. Stomach tube used in ileus for continuous stomach aspiration. The small metal tip (inset) allows the tube to be passed through the nose (see Figure III).

Drainage of the Stomach, Duodenum and Upper Part of the Jejunum by the Indwelling Stomach Tube.

The introduction of continuous gastric drainage by an indwelling stomach tube has marked a great advance in the treatment of paralytic ileus. Many designs of stomach tubes and apparatus for producing the suction have been described, notably by Levin[2] and Wangensteen.[3] A modification of their apparatus which is simple to prepare and efficient in practice will be described in detail. The stomach tube consists of a fine rubber tube one hundred and fifty centimetres in length and 0·4 centimetre in outside diameter. The end is fitted with a special metal tip fifteen millimetres in length and five millimetres in outside diameter, with one terminal and six lateral holes one millimetre in diameter, and a corrugated neck to receive the rubber tubing (see Figure II). The rubber tubing is perforated every five centimetres in the terminal thirty

centimetres. These perforations are one millimetre in diameter, and can easily be made by punching the tube right through transversely with a rubber dam punch.[1]

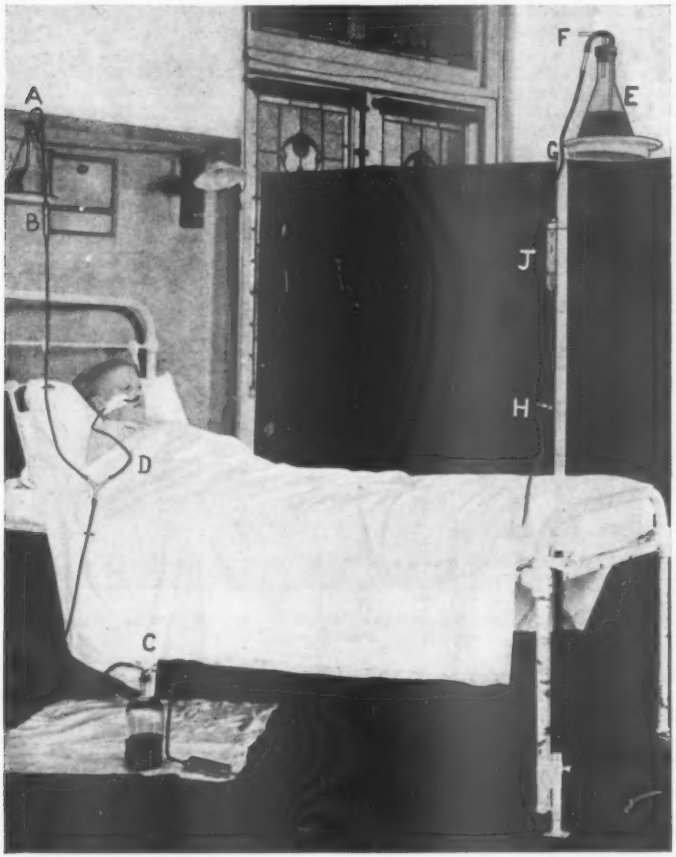

FIGURE III. Gastric aspiration and continuous intravenous infusion in a case of ileus. A, reservoir; B, tube and dropper; C, aspirating bottle and pump; D, stomach tube passed through the nose (see Figure II); E, intravenous reservoir; F, filling tube; G, delivery tube; H, regulating screw-clip; J, dropper.

This stomach tube is introduced through the nose in preference to the mouth, as it is easier for the patient to swallow, and when left in for any great length of time it is far less irritating to the mouth (see Figure III). If it hangs

[1] The special stomach tubes (see Figure II) may be obtained from leading wholesalers.

out of the corner of the mouth it evokes an excess flow of saliva and produces a constant desire to chew on it. The tube is passed through the nose until it reaches the pharynx, and then the patient drinks iced water slowly, when it will be found that the tube is gradually drawn down by the peristaltic waves in the œsophagus. The operator merely aids its passage into the nose, and should not thrust it in rapidly as this causes it to curl up in the mouth, producing a choking sensation.

When the tube has reached the stomach the gastric contents are aspirated by means of a large glass syringe, and when the stomach is emptied the patient drinks a further 500 cubic centimetres or more of saline solution, and this is immediately aspirated. This process is repeated till the aspirated fluid is practically clear. About thirty centimetres more of tubing is then fed through the nose in order to enable the tip of the tube to pass through the pylorus into the duodenum and upper part of the jejunum. The stomach tube is fixed to the face with a liberal amount of adhesive plaster, and then connected to a glass Y-tube as is shown in Figure III. The second limb of the Y-tube is connected to a graduated reservoir (A), via a dropper (B), and the third limb is attached to a graduated aspirating bottle and exhaust pump (C), such as has been designed

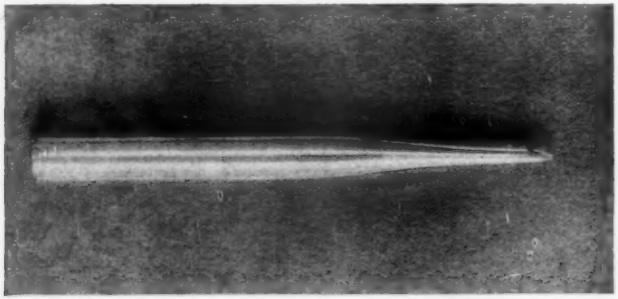

FIGURE IV. The glass cannula which is inserted into the vein for continuous intravenous infusion.

by Potain for the aspiration of pleural effusions. The reservoir (A) is filled with 20% glucose in twice normal solution of sodium chloride (1·8%) and 300 cubic centimetres are allowed to run slowly into the stomach at the end of each hour, the flow being regulated by a screw clip. If the patient is not sleeping this fluid should be partly substituted by fluid swallowed naturally, as this keeps the mouth clean and moist, and it also acts as a stimulus to digestion (Pavlov[6]). Glucose (20%) in half normal saline solution and flavoured with lemon is a suitable drink. Later barley water, Bengerized milk and eggs may be given to increase the caloric intake. The stomach is emptied by the aspirator at the end of every two hours or before this if the patient is nauseated or feeling uncomfortable. If the stomach tube becomes blocked and does not discharge its contents into the aspirating bottle, it should be disconnected from the Y-tube and aspiration should be carried out with the glass syringe. If the contents are still fæculent the stomach is again washed out as before.

By this means the stomach contents will gradually return to normal appearance, consistence and odour, even if they have previously been highly fæculent and dark brown or black in colour. The occurence of fæculent vomiting is no longer the herald of death.

This procedure is persisted with till it is found that the aspirated contents of the stomach are normal, more fluid is being put into the stomach than is being aspirated, all nausea has passed, the abdominal distension is subsiding

and the bowels are expelling flatus and fæces. In the ten cases treated by me the longest time in which gastric lavage had to be maintained was seven days, the shortest two days. This is in accordance with results reported by others. It is wise to withdraw the stomach tube and to pass it through the other nostril every three days, in order to avoid irritation of the nasal mucous membrane.

Accurate records are kept of the volume of the fluid intake and output by the mouth and stomach tube (see Figure V). It is usually found that for several days a far greater volume of fluid will be withdrawn than is given. There is a large negative balance. This must be made up by fluid injected rectally, subcutaneously and intravenously. Of these routes, intravenous infusion by the

FLUID BALANCE

		IN					OUT						
DATE	TIME	ORAL	TUBE	RECTAL	I.V.	TOTAL	TUBE	VOMIT	URINE	FAECES	SWEAT	TOTAL	BALANCE
4th	A.M. 1	-	150				400						
	2	-	150										
	3	150	-	500			200						
	4	150	-										
	5	200	-				300						
	6	100	50						150				
	7	150	100				150						
	8	100	50				250						
	9	150	-	500									
	10	150	-				100						
	11	150	-					NIL	90	NIL			+
	12	150	-		2000	2250	300				600	2540	110 c.c.
	P.M. 1												
	2												

FIGURE V. A suitable table for recording the intake and output of fluid in a case of ileus. At the end of each twelve-hour period the fluid balance is calculated. A positive balance should be maintained throughout.

continuous drip method is by far the most efficient. Before discussing this method in detail a few observations will be made on the rectal and subcutaneous routes.

Rectal Injection of Fluid.

In the early phases of ileus fluid may be administered *per rectum*, but once the condition is advanced it is debatable whether it is wise to inject large quantities of fluid into an atonic bowel. Sir Hugh Devine advocates the repeated use of gentle bowel washouts in order to encourage peristalsis and yet to avoid persistent over-distension of the bowel with fluid (personal communication). Moreover, it must be remembered that even under normal conditions it is doubtful whether any food is absorbed from the rectum. Even a simple and highly diffusible substance such as glcose is said to be unabsorbed by Corkill,[7] McNealy[8] and others. Thus other routes, notably the intravenous, must be used.

The Subcutaneous Injection of Fluid.

The subcutaneous route is simple and efficacious, but only small volumes of fluid can be injected for a restricted period of time. I do not employ it in established cases of ileus, preferring the intravenous route. Glucose, which is irritating to the tissues, should not be administered by this route, except possibly in very low concentration (glucose 1%).

The Intravenous Injection of Fluid.

When owing to negative balance it is necessary to supplement the amount of fluid given by mouth, the intravenous route is, in my experience, the most suitable. Unlimited volumes of water, salt and glucose can be injected over a prolonged period, even for as long as one or even two weeks, without any discomfort or danger to the patient. The technique employed by me has recently been described elsewhere,[9] so only a brief description of the procedure will be given.

The Apparatus.—The apparatus (see Figure III) consists of a graduated reservoir (E) made from a "Pyrex" glass Erlenmeyer flask of five litre capacity and fitted with a rubber cork pierced by two glass tubes (six millimetres),[1] one for filling and the other for delivery of the fluid. The reservoir is filled by means of a funnel and tubing which are connected to the filling tube (F) after the cork has been loosened to allow the air to escape. F is then loosely packed with cotton wool to prevent air contamination. Fluid syphons over from the reservoir into a delivery tube (G) constructed of ribbed rubber tubing such as is used for stethoscope tubing (size inside diameter four millimetres). The rate of flow is shown by a dropper (J), which consists of the barrel of a male glass syringe (capacity 60 cubic centimetres) fitted with a rubber cork which is pierced by a short glass tube tapered at the lower end. If the outside diameter of the tip of this tube is two millimetres, it will deliver approximately 150 cubic centimetres per hour if the flow is at the rate of 50 drops per minute. The rate of flow is regulated by a screw clip (H).

The delivery tube is fitted with a short glass cannula of standard bore, nine centimetres in length, and constructed with a tapering end and bevelled point (Figure IV).

Cleansing and Sterilizing the Apparatus.—The apparatus is thoroughly cleaned with distilled water and then wrapped in bundles after being covered with gauze and padded with wool to prevent breakage. These bundles are sterilized by autoclaving for thirty minutes at a pressure of fifteen pounds (ten minutes wet steam and then twenty minutes dry steam) and packed in a bag ready for use. Tap water contains toxic substances, and if these are allowed to dry on the surface of the apparatus they will be redissolved during the administration, and rigors, pyrexia and even collapse may be produced in the patient.

The Fluid.—It has been found that the most satisfactory fluid for intravenous administration is glucose (5%) in Ringer's solution. Buffering is produced by adding sufficient di-sodium hydrogen phosphate to give a pH value of 7·0. When purified dextrose (glucose), specially prepared for intravenous work and corresponding to the British Pharmacopœia standard, 1932, is used in conjunction with the specially distilled water, it is found that approximately 1·18 grammes of the buffer are required for one litre of a 5% glucose solution. A simple polychrome indicator is suitable for determining the correct pH. Finally, sterilization is carried out by boiling for twenty minutes in an Erlenmeyer flask, the mouth of which is then plugged with sterile cotton wool. Solutions should be stored in a cool place and used within forty-eight hours of preparation. The solution is made up with water which has been filtered through a Jeffrey filter candle and then distilled in an all-glass still fitted with a Young's glass fractionating tube containing five baffles which prevent toxic substances from passing over into the distillate (Cooper and Haldane[10]).

[1] These types of glass and rubber tubing are of standard size, and are used in all the various procedures described in this paper. "Pyrex" glass is used throughout.

The Technique of Intravenous Injection.—The apparatus is first assembled, filled and supported on a stand three feet above the level of the bed (Figure III). Fluid is sucked over from the reservoir (E) into the dropper (J) by removing the cork from the dropper, filling the dropper and its attached tubing with

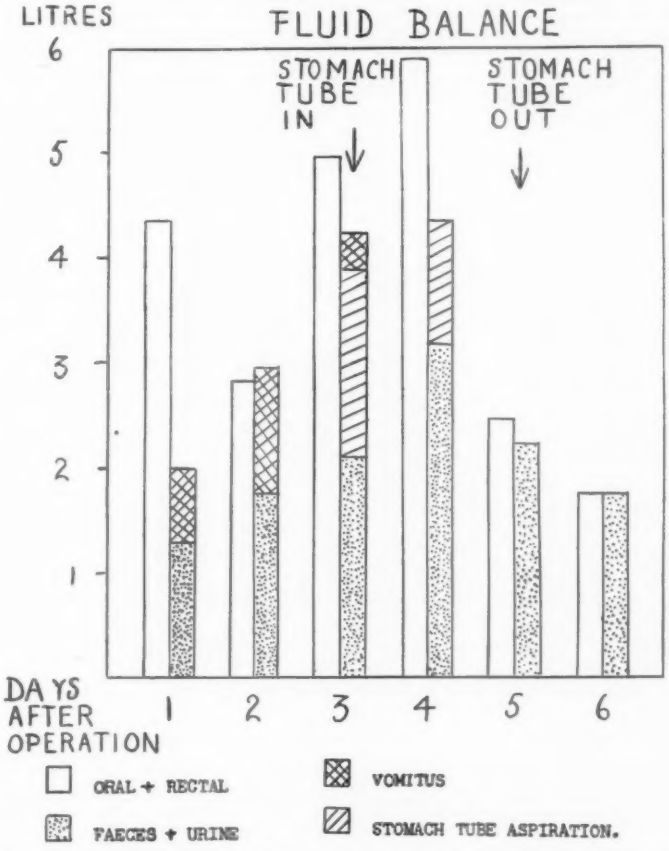

FIGURE VI. The fluid balance in a case of ileus following the division of a peritoneal band for intestinal obstruction. Treatment by stomach aspiration was begun early and adequate fluid replacement was obtained by the oral and rectal routes.

the glucose-Ringer's solution and then replacing the cork. Then as the level of fluid in the dropper falls it sucks fluid over from the reservoir till it eventually enters the dropper. By this time a suitable airlock has been established in the dropper. Finally the screw clip (H) is closed. A suitable vein is then exposed. The internal saphenous vein immediately above the internal malleolus has been

found by me to be the most suitable, as it is constant in position, easy to expose and allows the various nursing procedures to be carried out with ease.[11] If a vein in the arm is used, it does not allow a restless patient complete freedom of movement of his arms, and he is continually aware that the infusion is taking place. After the cannula has been thrust up the vein till it engages firmly in the lumen and then tied in position with a ligature of catgut (size 00), the cannula and a portion of the attached tubing are secured to the leg by transverse

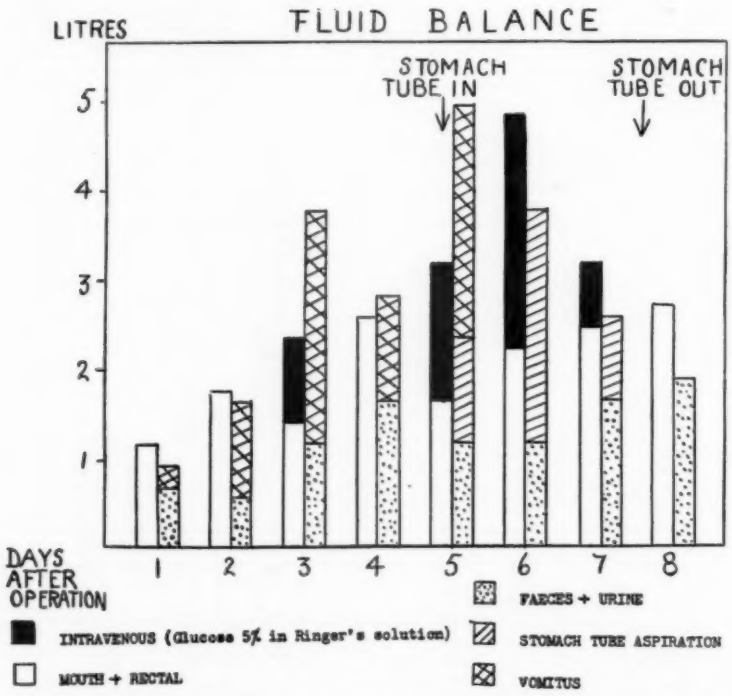

FIGURE VII. The fluid balance in a case of ileus following on the removal of a suppurating appendix. An intravenous infusion of one litre given on the third day was inadequate. Continuous gastric drainage and continuous intravenous infusion were begun at midday on the fifth day. There was no further vomiting and the patient slowly improved.

strips of strapping. Finally, the whole foot is covered with a liberal amount of cotton wool which is held in position by a firm bandage. The only restriction placed on the movement of the leg consists of connecting the big toe to the foot of the bed by a length of bandage a few centimetres shorter than the free length of rubber tubing which leads to the cannula. This hobbling bandage is attached to the big toe by means of adhesive strapping.

Volume of Fluid Injected Intravenously.—Intravenous administration is indicated if sufficient amount of fluid, salt and food cannot be administered by the more simple methods, bearing in mind the inefficiency of the rectal route.

The volume of fluid to be given is estimated first by studying the clinical condition of the patient, especially the thirst, sunken eyes, tongue, volume and frequency of the pulse, and the volume of urine passed; and, secondly, by keeping a careful record of all the fluids which pass in and out of the body by the various routes. Accurate details are kept of the volume given and the volume lost by the vomitus, stomach aspiration, urine and fæces. In cold weather about 500 cubic centimetres are allowed for sweating and expired moisture and in hot weather relatively greater volumes. Figure V shows a suitable table which could be kept by the nurse in charge of the case. At the end of each twelve hours the chart is balanced and a *plus* or *minus* figure is arrived at. When the intravenous administration is first begun the patient will be dehydrated from excessive vomiting, so it will be necessary to produce a large positive balance of, say, two to three litres in the first twenty-four hours. As the signs of dehydration pass, a smaller positive balance should be produced—about 500 to 1,000 cubic centimetres in each twenty-four hour period. From practical experience it has been found that in a patient weighing 60 kilograms it is necessary to give approximately four litres of fluid in the first twenty-four hours and three litres on each of the ensuing days.

The beneficent effects produced in the patient by the massive intravenous administration are an improvement in his anxious and restless appearance, the pulse becomes slower and of better volume, the blood pressure rises, the cyanosis fades, thirst is less intense and sleep is easier to produce. The appearance of the tongue has always been regarded as a useful guide to the degree of dehydration which is present. Although a dry tongue usually signifies dehydration, the converse is not always true, for I have frequently observed quite a moist tongue in the presence of indisputable dehydration. Too much reliance should not be placed in this single sign.

Figures VI and VII show the effects of continuous gastric aspiration and fluid replacement on the fluid balance in two cases of post-operative ileus.

Figure VI represents a mild case of ileus which developed after an operation for the relief of subacute intestinal obstruction from a band. Treatment was instituted early, and vomiting ceased immediately the stomach tube was passed. Fluid replacement was satisfactorily carried out by the oral and rectal routes.

Figure VII shows the excessive fluid loss in a more severe case of paralytic ileus following on the operation of appendicectomy for an acute suppurative appendicitis. Here it was essential to inject intravenous fluid to maintain a positive balance. This was one of the first cases that I treated by the indwelling stomach tube, and it will be seen from Figure VII that on the third day the relatively small volume of fluid (one litre) injected and the failure to insert the stomach tube proved unsatisfactory. Vomiting persisted, and there was a negative fluid balance. At midday on the fifth day the stomach tube was passed, and the continuous intravenous drip was begun with very gratifying results.

Summary.

1. The signs and symptoms of ileus are recorded.

2. The normal distribution and movement of water in the body are discussed with special reference to the gastro-intestinal circulation.

3. The treatment of ileus by aspiration with the indwelling stomach tube and by fluid replacement by the continuous intravenous drip method is discussed.

4. The necessity of maintaining a positive fluid balance is emphasized.

Acknowledgement.

I beg to express my sincere thanks to Dr. Charles Kellaway, Director of the Walter and Eliza Hall Institute, Sir Hugh Devine and Sir Alan Newton for their great help and encouragement with this investigation. Miss Beryl Splatt, biochemist of the Royal Melbourne Hospital, has given much valuable advice. I am deeply indebted to the honorary medical and surgical staffs of the hospital for generously placing clinical material at my disposal.

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Case Reports.

VOLVULUS OF A MECKEL'S DIVERTICULUM.

By H. R. G. Poate,

Sydney.

E.R., a male, aged thirty-eight years, was admitted to hospital with symptoms of an acute abdominal emergency of some thirty-four hours' duration.

He was a returned soldier, pensioned in 1922 for pulmonary tuberculosis (after an old tuberculin test); but the condition appears to have been arrested for many years, as an X ray examination in July, 1934, showed evidence only of chronic bronchitic changes in both lungs.

Figure I. Meckel's diverticulum as removed. Note twisted and narrow neck as contrasted with the unusual size of the diverticulum.

There was nothing of note in his family history, but in his personal history it was remarked that he was mentally dull, of slovenly habits, a moderate smoker and practically a teetotaller. He had a cough which was largely "functional" and easily controlled, there being no sputum.

In 1926 he was operated on in one of the larger metropolitan hospitals for appendicitis, his symptoms suggesting a chronic condition. Symptoms recurred after the operation with occasional acute exacerbations. Several opaque meal X ray examinations of the alimentary tract revealed no abnormality.

The present illness commenced about midnight with acute generalized abdominal pain, colicky in nature, which doubled him up at times. After some hours the pain seemed to settle low down in the right side of the abdomen and vomiting ensued twenty-four hours after the onset of the illness. As the pain was becoming worse, he was sent to hospital thirty-four hours after the trouble first began. His bowels had been well opened twice in this period.

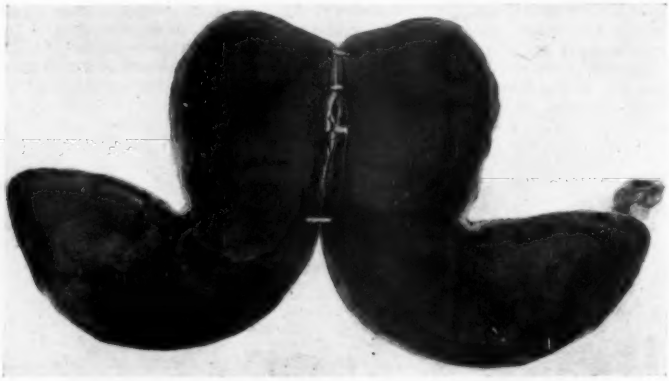

FIGURE II. The diverticulum in section filled with gelatinous material.

Upon examination at 2 p.m. his tongue was furred, but moist. His temperature was 36·7° C. (98° F.), and his pulse rate was 96 in the minute. His heart and chest were clear, except for a few crepitations at the base of the right lung.

The abdomen was held rigidly, and extreme tenderness was present in the epigastric, right hypochondriac and iliac regions. There was a large left inguinal hernia which was reducible, but manipulation increased his pain. In the afternoon two enemata were administered, but returned clear of fæcal content. A leucocyte count revealed 12,300 cells per cubic millimetre. At 6 p.m. another enema was given and again was returned clear. He had no vomiting, but the abdomen was rigid and tender. The temperature was still 36·7° C. (98° F.), but the pulse rate was 120. Rectal examination revealed no abnormality. He was given a hypodermic injection of morphine and seemed to settle down.

At 5 a.m. next day a milk and molasses enema was returned clear. The abdominal condition was much the same, but his temperature was 38·3° C. (101° F.), and his pulse rate was 104; at 9 a.m. the leucocyte count was 13,000 cells per cubic millimetre.

He was then seen by me for the first time, and because of the previous history of chronic dyspepsia, the acute onset and the board-like rigidity, the condition was thought most likely to be a leaking duodenal ulcer which demanded immediate operation.

A right paramedian supra-umbilical incision was made, but the stomach, duodenum and gall-bladder appeared normal. When I passed my hand into the

lower part of the abdomen a large hard mass was palpable in the right iliac fossa and in the right side of the pelvis.

The original incision was closed, and another incision was made in the lower part of the abdomen. At first it was difficult to determine what the condition was, as several coils of small intestine were adherent over the area; but when these were freed a very large gangrenous mass became evident. When it was lifted from the lower part of the pelvis it was found to extend upwards, and an attachment to the lower portion of the ileum was found.

The condition was then recognized as a Meckel's diverticulum which had become twisted round some three or four times on its base; the base was narrowed to such an extent that crushing with forceps and ligature were the only procedure necessary to remove it. It was completely gangrenous and contained a quantity of red gelatinous material.

Despite the long delay in instituting operative treatment (sixty hours) and the intense inflammatory reaction, the patient made an uneventful convalescence.

Acknowledgements.

I am indebed to Dr. Pardy, of the Prince of Wales Hospital, for the clinical notes; to the Deputy Commissioner of Repatriation for permission to publish the case; and to Professor Dew, of the Department of Surgery, University of Sydney, for the photographs which are reproduced.

GENERALIZED CARCINOGENESIS IN AN ADULT KIDNEY.

By R. J. SILVERTON,
Sydney,
AND
EMERITUS PROFESSOR D. A. WELSH,
Sydney.

Clinical Report.
(R. J. Silverton.)

A FEMALE patient, S.D., aged sixty-five years, was referred to me at the Royal North Shore Hospital, Sydney, on October 10, 1935, complaining of fairly continuous, severe and sometimes agonizing pain in the left loin for the previous

FIGURE I. Mrs. S.D. Left retrograde ureterogram. The sodium iodide solution will not enter the left renal pelvis. The renal shadow itself is seen to be somewhat enlarged, and is more rounded in shape than is normal.

FIGURE II. (Natural size.) Complete hemisection of the carcinomatous kidney shows (i) more or less uniform enlargement of the whole kidney, suggesting generalized carcinogenesis from renal epithelium *in situ*; (ii) general pallor of the cut surface, with occasional areas of hæmorrhage and necrosis; (iii) invasion of the renal pelvis, with obliteration of the calyces and dilatation of the ureter.

ten weeks. For about twelve weeks she had been passing clots of bright blood from the bladder. No subjective vesical symptoms were present. The patient looked moderately well. A firm rounded mass was palpable in the left renal area, both anteriorly and in the loin. It was slightly tender on pressure. The urine contained many red cells, an occasional pus cell, but no microorganisms. A plain radiogram was clear, except that the left renal shadow was more prominent and slightly larger than that on the right side.

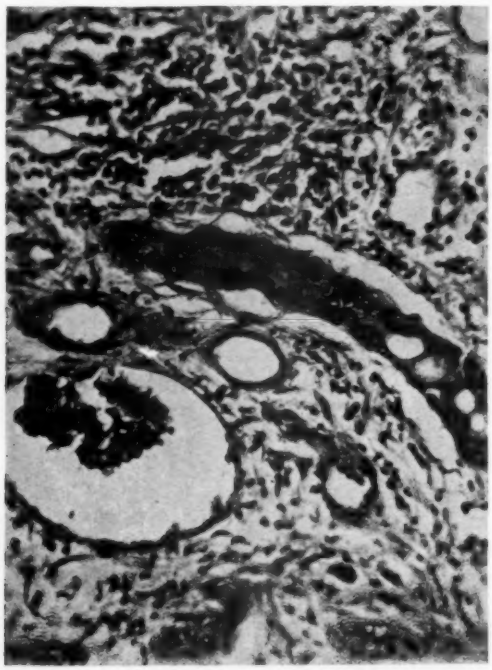

FIGURE III. (×200.) Advancing margin of carcinoma in surviving subcapsular cortex shows (i) a cortical tubule in process of carcinoma *in situ*, (ii) a glomerulus not yet involved, (iii) a defensive zone of lymphocytes separating the cancerous from the non-cancerous cortex (which does not appear in the field).

Cystoscopy revealed a good indigo-carmine excretion on the right side, but no efflux was seen on the left side. A catheter was passed up the left ureter quite gently, and lubricated with "Vaseline" as is my routine, but when it reached the kidney bright blood entered the catheter, a little escaping at the other end, but most of it clotting rather quickly in the catheter. This rendered retrograde pyelography impossible on this occasion.

A few days later excretion urography revealed a normal right kidney, but no excretion at all showed up on the left side. Cystoscopy was repeated and a catheter was passed up the ureter not quite to the kidney and very gently to avoid hæmorrhage. A pyelo-ureterogram was made. Figure I shows the sodium iodide

going up as far as the left uretero-pelvic junction, but not entering the kidney pelvis.

The left kidney was explored and found to be moderately enlarged, rounded and extremely tense, and nephrectomy was decided on. During the freeing of the kidney the suprarenal gland was found to be moderately adherent to the upper pole, so was carefully removed together with the kidney.

Section of the kidney shows a mass uniformly yellowish-grey, with a few cavities into which hæmorrhage has taken place. Neoplastic tissue has completely

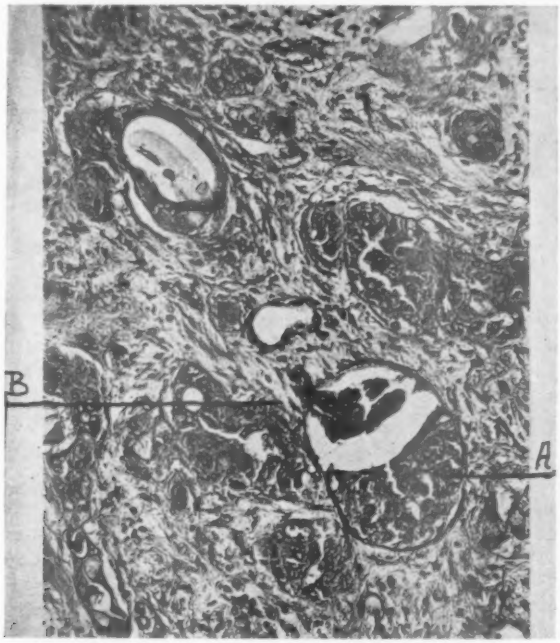

FIGURE IV. (×160.) At a deeper level in superficial cortex the carcinoma is more developed, still taking origin from secreting tubules, but now invasive, and also developing in a glomerulus from the epithelium lining Bowman's capsule, but only as carcinoma *in situ* not yet invasive. A mass of cancer cells at A (obviously derived from the epithelium lining Bowman's capsule) occupies about half the space between the capsule and the capillary tuft (which is collapsed against the afferent and efferent vessels) well seen at B.

replaced renal tissue; further, it fills up and replaces the entire pelvi-calycine system, thus explaining the appearance in the attempt at retrograde pyelography. The section has an entirely different appearance from that seen in the usual hypernephroma with its diverse areas of brilliant colouring (see Figure II). Moreover, in the typical hypernephroma, even when advanced, a fair amount of renal tissue is still left. In this case the neoplasm is obviously renal in origin and has evidently, right from the beginning, affected the whole organ simultaneously. In the typical hypernephroma, however, one gets the impression of

the growth having commenced in one portion of the organ, or even conceivably outside the kidney, destroying it gradually as it invades the organ from the point of commencement.

The patient was very well when she left hospital a few weeks after operation; but, as will be seen from Professor Welsh's notes which follow, invasion of the suprarenal gland had already commenced, and no doubt metastases were already present in distant parts when the kidney was removed. I received news that the patient died about seven months later.

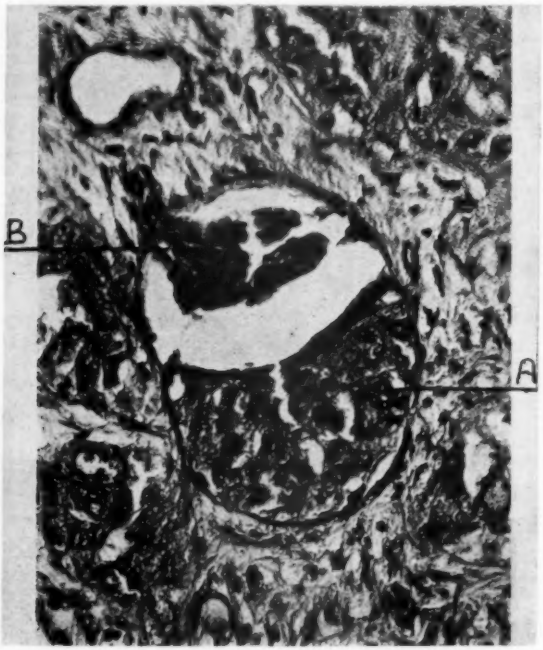

FIGURE V. (×300.) The same carcinomatous glomerulus, with the same lettering as in Figure III, under higher magnification, illustrates progressive carcinogenesis within Bowman's capsule. Compare and contrast this neoplastic reaction of the glomerulus with the inflammatory reaction in glomerulitis shown in Figure VI.

Pathological Report.

(Emeritus Professor D. A. Welsh.)

In order to understand the complicated pathology of this tumour, one must realize that a carcinoma may spread in two fundamentally different ways. First and always, there is the recognized spread by invasion, which is the essence of malignancy. The malignant cells acquire the power of leaving their site of primary origin and of forcing their way through adjacent tissue to form masses of cancer cells in the tissues invaded (the local invasion). In addition, the

malignant cells may invade lymph or blood vessels and be carried to distant parts where similar masses of cancer cells may grow (the general invasion).

Secondly, there is also a spread by contagion, which is not generally recognized, but which can often be demonstrated at the advancing margin of a carcinoma. Here the cancer extends by the healthy epithelial cells, in immediate contact with the cancer cells, becoming progressively cancerous while they still occupy their proper place in the normal architecture of the tissue to which they belong—progressive carcinogenesis *in situ* or nascent cancer—as if

FIGURE VI. (×300.) A glomerulus in subacute glomerulo-interstitial nephritis, following measles in a girl of ten years, illustrates (at A) the excessive inflammatory proliferation of the epithelium lining Bowman's capsule. The glomerular tuft (at B) is swollen and lobulated, its endothelium also proliferating. The secreting tubules are lined by degraded cubical epithelium

some malignant influence has been handed on from the marginal cancer cells to adjacent non-cancerous cells until they too become transformed into cancer cells. Then these new-born cancer cells develop the two attributes of malignancy which they had derived from pre-existing cancer cells, (*a*) the power of invasion and (*b*) the power of passing on to the next adjacent epithelium the "something" which changes non-cancer into cancer, and so the spread by contagion proceeds. I have discussed these processes of "contact spread" and "progressive carcinogenesis *in situ*" in more detail in *The Journal of the Cancer Research Committee of the University of Sydney*, 1935 and 1936.

Now the interesting feature of this primary carcinoma of an adult kidney is that it exhibits to perfection both these modes of cancer spread. All the histopathological evidence points to the fact that the carcinoma originated in the secreting tubules of the cortex, and that from that primary origin there developed (i) a spreading invasive cancer throughout the kidney and beyond it, and (ii) an extension of the cancer by progressive carcinogenesis *in situ* to glomeruli in the cortex and to collecting tubules in the medulla.

Progressive Carcinogenesis in Situ.

The invasive growth is obvious, and I shall refer to it again. But the extension of the cancer by progressive carcinogenesis *in situ* is no less obvious, if one knows where and how to look for it. I had noted early that a narrow

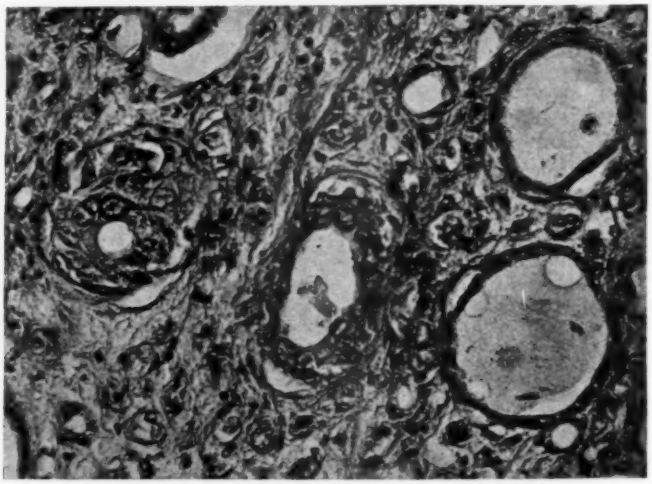

FIGURE VII. (×300.) In the surviving medulla, bordering the renal pelvis, progressive carcinogenesis *in situ* is developing within collecting tubules.

strip of superficial cortex was not yet involved in the cancer process and that, within the advancing margin of the cancer there, most of the secreting tubules were already cancerous and invasive, but only a few glomeruli (roughly speaking, about one in ten) were cancerous, and then only cancerous *in situ*, not yet invasive. Later, when I had examined a number of serial sections involving a wedge of medulla bordering on the renal pelvis, I noticed a similar process of carcinogenesis developing within the collecting tubules there. It then became obvious that many of these collecting tubules in the medulla and some of the more superficial glomeruli were in an earlier phase of carcinogenesis than were most of the cortical tubules. It followed that, starting in the deeper cortical tubules, the cancer had spread both up and down the tubular system of the kidney to involve representatives of all its epithelial elements, including glomeruli and collecting tubules, not by an invasive process, but by a process of progressive carcinogenesis *in situ*, whatever the explanation of that may be.

In the affected glomeruli the epithelium in immediate contact with the cancerous tubular epithelium, that is, the epithelium lining Bowman's capsule, was undergoing progressive carcinogenesis to form a mass of cancer cells between

the capsule and the capillary tuft. The epithelium covering the tuft was not found to be involved, but the tuft was collapsed against its root by some fluid effusion.

While the epithelium lining Bowman's capsule readily undergoes an inflammatory reaction with excessive proliferation in certain forms of glomerulitis, this is the first occasion on which I have seen that epithelium undergoing a neoplastic reaction. Ewing, however, in his invaluable work on "Neoplastic Diseases", third edition, page 794, gives these references: "Sharkey believed he could trace the origin of his tumor to the glomerular epithelium. Beneke also has observed tumor-like hyperplasia of glomerular epithelium."

In the collecting tubules of the medulla progressive carcinogenesis *in situ*

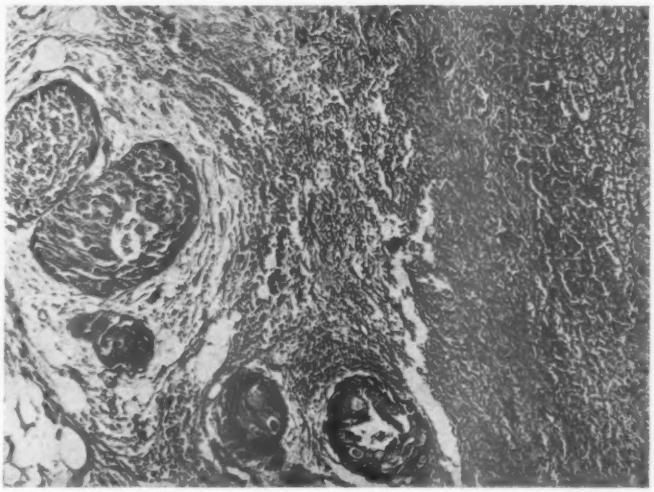

FIGURE VIII. (×80.) An invasive phase of the cortical carcinoma is attacking the adrenal through its adherent capsule. The large deeply staining cells and large cell masses of the renal carcinoma (in left of field) contrast with the smaller lightly staining cells of the adrenal cortex (in right of field).

begins by the gradual enlargement of their epithelial cells with increasing depth of staining both in cytoplasm and in nucleus—the pre-cancerous stage of nascent cancer. This reaction develops until a small mass of cancer cells is formed often at one part of the circumference, giving an eccentric thickening of the epithelial lining. Ultimately the tubule becomes blocked by the growing cancer cells.

All these facts in regard to progressive carcinogenesis *in situ*, as it appears in glomeruli and in tubules, are illustrated in the accompanying photomicrographs.

The Invasive Growth of the Renal Carcinoma.

When the carcinoma first appears in a tubule, the cancer cells tend to fill up the tubule and to obliterate the lumen, and later to distend and distort the tubule. Soon the cancer cells break through the wall of the tubule and begin to invade the connective tissue outside. At first this invasion takes the form of a diffuse local scattering of the cancer cells, which later grow into separate alveolar masses, with little tendency to reproduce a lumen. More often a false

lumen appears from degeneration of some cancer cells in the alveolar mass. Hence the type of cancer is more alveolar than adenocarcinomatous.

This invasive growth is found widespread throughout the kidney; only small areas of cortex and of medulla have escaped. The growth has also passed beyond the kidney to cause a massive invasion of the renal pelvis with obliteration of the calyces. It has passed through the renal capsule to invade the adherent adrenal. It has formed a mass of invasive growth projecting from the renal

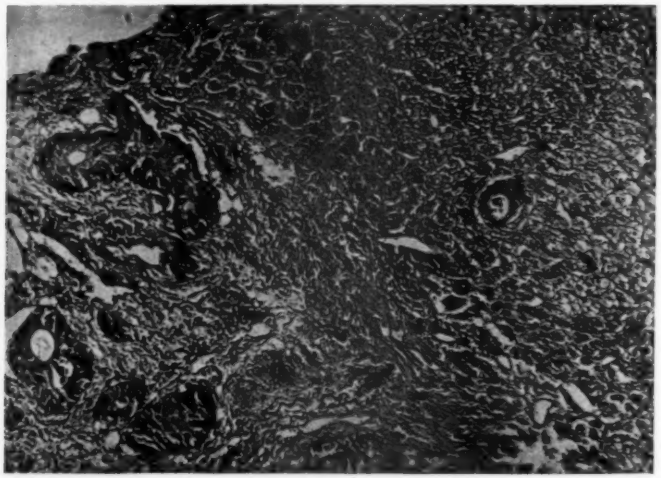

FIGURE IX. (×160.) Adherent adrenal in right of field is being invaded by small cell masses of cancer penetrating the two adherent capsules from the renal carcinoma in left of field.

pelvis beyond the kidney and overlapping the upper pole. In that mass of cancerous tissue beyond the kidney perivascular lymph spaces were found to be extensively invaded by cancer cells, so that the generalized dissemination was already on the way.[1]

Acknowledgements.

We are indebted to the medical artist, Mr. Woodward Smith, for the excellence of all the photographs, and to the Cancer Research Committee for providing them.

[1] A more detailed account of the pathology of this remarkable carcinoma, with additional photomicrographs, has been published in *The Journal of the Cancer Research Committee of the University of Sydney*, under the heading "Carcinoma Developing from the Tubular and Glomerular Epithelium of an Adult Kidney", July, 1937.

The Australian and New Zealand Journal of Surgery.

All articles submitted for publication in this journal must be typewritten and double or treble spacing should be used. Each article should conclude with a brief summary and statement of conclusions. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without any abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal together with that of the journal in which the abstract has appeared, should be given, with full date in each instance.

When illustrations are required, good photographic prints on glossy gaslight paper should be submitted. Line drawings, charts, graphs and so forth should be drawn on thick white paper in India ink. Authors who are not accustomed to prepare drawings of this kind, are invited to seek the advice of the Editor if they are in any doubt as to the correct procedure. Skiagrams can be reproduced satisfactorily only if good prints or negatives are available.

VOL. VII. APRIL, 1938. No. 4.

THE PRECLINICAL SUBJECTS IN MEDICAL TRAINING.

AT one time a patient was regarded as fit who did not return to the doctor for further treatment. Conversely, the individual who avoided the practitioner was considered in good health. The profession in its complacency never included the possibility that the patient might prefer to put up with his or her disability, whether physical pain, morphological or functional deformity or impaired æsthetic appearance, rather than seek medical advice. Later the profession introduced more objective standards of fitness. Medical men realized that they could not restore people to their normal function unless they knew the normal function, what it was and how it was maintained. With this realization of the fundamental importance to the practitioner of accurate anatomical and physiological knowledge, the apprenticeship system disappeared and the preclinical training was introduced. The student was now taught physiology and anatomy as a part of his training before ever meeting with any patients; simultaneously he became devoted to the study of

chemistry, physics and biology, although not in so demarcated a manner as they exist at the present day.

When Vesalius taught anatomy, he taught to his contemporaries and students the results of his personal investigations. Again, John Hunter taught anatomy and surgery in the sense that he imparted to his fellow students and surgeons the results of his own researches, and did not allow himself in his teachings to be hide bound by tradition. Again, in our own era, Sherrington at Oxford has taught physiology in the sense that he has conducted original investigations into the functions of the spinal cord and brain, and has imparted his results to his fellow workers in his laboratory and to his contemporaries throughout the world at meetings. Many Australian and New Zealand workers privileged to hear and work with Sherrington retain memories, not of a fluent lecturer or an able exponent of the slide and lantern technique, but of the inspired interpreter of the nervous system as revealed by his experiments and those of the school he built up.

Cardinal Newman was responsible for founding University College, Dublin (later to grow into the National University of Ireland), and he stated that the primary function of a university was to teach. This word "teach" is too often taken to mean solely the conduct of classes and demonstrations accompanied by a high percentage of examination passes without any or only a perfunctory performance of original work, without any gift of new knowledge being made by the teacher to his contemporaries and successors. This is not teaching in the truest and highest sense.

The prosecution of original investigation by a body of investigators, the continuous satisfying of "the devil of scientific curiosity" as to the quality of truth in knowledge, is nowadays dignified by the title of research. This endless investigation resulting in the widening of the bounds of knowledge, the accompanying communal interchange of ideas and the discussions between fellow workers, between master and student—these are the qualities which distinguish the university from the technical school, the polytechnic and the coaching class. The great universities of the past, which, though still existing, are no longer places of pilgrimage for the curious student, have lost their headship because they have lost sight of this ideal. In the search for knowledge and truth we are as a boat rowing upstream: we can never rest our oars, for we are at once washed down to mediocrity. Those universities of our era, both old and new, which are the places we look forward to visiting in our sabbatical years, are those which are continuously

encouraging research as well as doing their routine teaching, which have something new to teach us. What has been written here applies to all subjects in all faculties, but it is of particular importance to the physician and the surgeon because he has clinical material passing through his hands day by day. His duty is twofold—to cure his patient, and to learn from each case something which will improve the prevention and cure the condition exhibited. To achieve this he must be equipped with that training which will enable him to build his experience into a realistic philosophy of medical science.

The attitude which regards teaching as merely routine instruction in determined knowledge, and this as the primary function of a university medical man, arises from two causes. First, one can make a living without doing any original work because too often governing bodies judge the excellence of a university by its examination results, and tend to discourage financial expenditure on works of research. Secondly, there is a tendency for physicians and surgeons to love what is called the "practical". If no immediate application to the patient arises out of the new knowledge, then the work is not "practical". This is because before any work can be applied to the patient the basic physiological and anatomical knowledge must be worked out first—after that the application which restores the physiological distortion (pathological) to the physiological normal will follow with ease. It behoves us, therefore, to teach in the highest sense of the word, and to train our young men and women in the results of investigation (the known corpus of anatomy and physiology), in the methods of original research and in the conduct of these same researches, so that our students may carry on in the laboratory and the ward the work which has been handed on from Vesalius and Leonardo, through Harvey, Laennec, Ludwig, Bernard, Pasteur and Lister, Sherrington and Hopkins, and Lewis and Cushing, and those others of our contemporaries who are increasing the sum total of knowledge so as to make it of use to mankind.

A. ST. GEORGE HUGGETT.

Surgery in Other Countries.

[In this column will be published short résumés of articles likely to be of practical value from Journals published in other countries and not readily accessible to surgeons in Australia and New Zealand.]

A Simple Method of Guiding the Nail in Extraarticular Osteosynthesis of the Femoral Neck Fractures with a New Instrument.

José Valls and E. H. Lagomarsino: "*Technik und neues Führungsinstrument für die extraartikuläre Nagelung von Schenkelhalsbrüchen*", *Zentralblatt für Chirurgie*, 1937, Number 20, page 1170.

Report of discussion at meeting of Society of Surgeons, Vienna, 1937, by **Böhler, Felsenreich, Jerusalem, Schnek** and others. *Ibidem*, 1937, Number 16, page 944.

E. Gold: "*Schenkelhalsnagelung nach der Argentinischen methode* (Valls)", *ibidem*, 1937, Number 24, page 1411.

P. Fuchsig: "*Zur Technik der Schenkelhalsnagelung*", *ibidem*, 1937, Number 27, page 1596.

It is evident from the international literature that the Smith-Petersen nail is being used very extensively. It is equally true that the original intraarticular method has been abandoned by nearly every authority (E. Hey Groves, Smith-Petersen, Boppe, Lelio Zeno, and, according to reports, Watson-Jones and others). The many poor results still obtained by the newer method of extraarticular nailing by some surgeons are generally due to avoidable mistakes, such as bad selection of cases, poor reposition, inaccurate nailing or failure to insert the nail in an ideal place and, finally, too early weight-bearing (under three to six months). The Argentine surgeons Valls and Lagomarsino have described a guiding instrument for the nail based on a different principle to others. They acknowledge that it is really inspired by the famous "cannon" of Delbet, invented many years ago, and the later instruments of Brocq and Dulot, Sterling Bunnel (United States of America), and to this list may be added those of E. Hey Groves, Schmidt and others, recently described. The unique instrument of Eric Lloyd (London) and all other guiding instruments obtain the aim from the lateral aspect of the femur; but with the Valls instrument the direction is obtained from the *caput femoris*. It was used originally for intraarticular nailing. The instrument consists of a rigid three-sided, but adjustable, frame constituting with the fourth side (the Smith-Petersen nail) a sloping parallelogram. Note that the spike for penetrating the femur is about half the diameter of an average *caput femoris* or 2·2 centimetres shorter than the opposite side of the parallelogram which holds the nail. The obliquity of these two sides of the instrument is to provide that the spike is oblique and so will pass under the femoral artery and not into it during passage of the spike which is dangerously near it (Figures III and IV). The instrument has been designed in order to make available to all surgeons a simple method of extraarticular nailing, which, according to Valls, requires only fifteen minutes. Unfortunately, a most careful observation of certain most minute technical requirements is essential, and certain criticisms directed towards improving the method have been made (see below). It has been rather extensively demonstrated and used by Valls and Viennese surgeons, to whom he showed the method during his recent visit there. The interest was such that Odelga (Vienna) is now advertising and supplying it. The instrument (Figure I) has a director for the nail (or a wire, according to some models) on one side and the spike on the other. The spike is thrust into the *caput femoris*, and for this important

manœuvre it is separated at the cross bar from the nail guiding arm (Figure II). These are later joined up and thus an adjustment for varying lengths of nail is always permitted by the sliding cross bar, also a measuring scale is available for determining the length of nail required. Valls's technique is as follows: Reduction is carried out, frontal and lateral X ray control pictures are taken; but

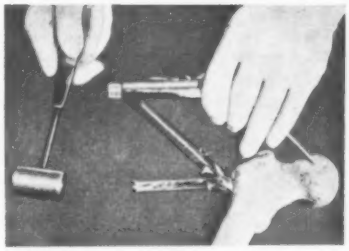

FIGURE I.

FIGURE II.

no more lateral X ray examinations are made if the reduction is correct, because they are unnecessary on account of the instrument. Fuchsig, who saw Valls demonstrate the instrument and has used it himself fourteen times, disagrees with Valls on this point. In Fuchsig's first four cases the nail was in a very good position in only three cases, although the patient recovered satisfactorily. For the next ten cases he used Valls's instrument, but modified it to hold a Kirschner wire (which Valls had abandoned as unnecessary), the position of which was controlled by lateral in addition to frontal X ray pictures. Fuchsig thinks that if these two factors (that is, the wire guide and lateral X ray pictures) which Valls thinks are unnecessary are omitted, the method does not guarantee the exactitude which the nailing requires. After the control X ray examination of the reduction has been made and the result is satisfactory, two lead markers are placed on the skin with adhesive

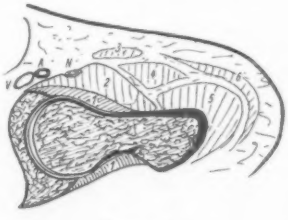

FIGURE III.

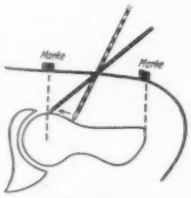

FIGURE IV.

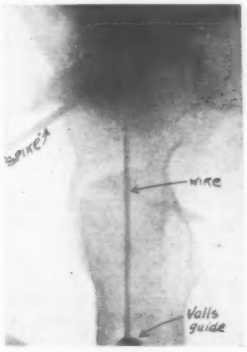

FIGURE V.

plaster, one in the region of the head (one centimetre below and outside the angle between Poupart's ligament and the femoral artery) and the other outside the base of the trochanter. Another frontal X ray examination is made, but it appears that this placing of the markers could have been done in conjunction with the first X ray examination (Figure VI). The sharp point of the spike is now thrust under the femoral nerve and artery in an oblique fashion (Figure II),

but the finger throughout should be on the femoral artery. It is better to thrust the obliquely set spike onto the neck some distance outside the *caput femoris*, over which the femoral artery passes (Figures III and IV) as indicated by the dotted line showing the direction of the spike in Figure IV, which shows it impinging upon the femoral neck. Thence it is gently slid along medially until the expanding junction of the head with the neck is felt. Then it is thrust into the head itself, and engaged there. The portion of the instrument holding the spike is then held by hand, whilst the frontal X ray examination is made, as shown in Figure II. The lateral incision is then made, and the other side of the parallelogram united by the sliding cross bar to the part which holds the spike. The nail is adjusted in its guide so that it will penetrate the lateral aspect of the femur just below the base of the great trochanter. If the X ray examination shows (Figure VII) that the spike is not centrally placed in the head, it can be moved slightly and the lead markers can be used as a guide. When this adjustment has been made it may not be necessary for a further X ray frontal examination to be made; but everyone will not agree to this. The lateral X ray picture reproduced in Valls's paper (but not here) does not show the nail in an ideal position,

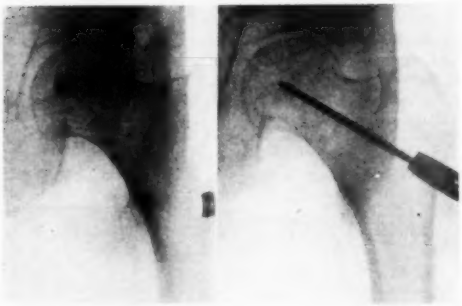

FIGURE VI.

FIGURE VII.

FIGURE VIII.

although it was satisfactory because osseous union resulted. Valls reported thirty cases, nine intraarticular and twenty-one extraarticular. Of the first twenty cases (over six months), 75% united by osseous union amongst the intraarticular group (one death from infection and one pseudarthrosis) and 100% in the extraarticular group. Summing up the question of osteosynthesis, he states: "We believe that many of the bad results published are due to inexact reposition and bad position of the nail." Felsenreich, at Denk's Clinic, where, since December, 1932, he has operated in 139 cases, stated that the instrument should be used with wires, because otherwise the *caput femoris* might rotate during the insertion of the nail, which he has observed in nine cases. Although he has used every type of guiding apparatus that he has seen described, he has always relied on none. However, this new apparatus is to be commended, and he has altered it so that it will take both wires and the nail. Böhler saw Valls operate both quickly and satisfactorily, but thought that the guiding wires should be used because a misdirected wire is so easy to remove. But if, as judged by Valls's instrument, a nail seems in a doubtfully good direction before it has been driven in, and if it still seems so when driven in, then the surgeon would hesitate to remove it because so much damage to the bone results. Lateral X ray examination should not be omitted.

In conclusion, it may be stated that the important Viennese surgeons interested in this subject all spoke favourably of the instrument and are using it; but unanimously agreed that it should be used in conjunction with guide wires and lateral as well as frontal control X ray examinations. Note in Figure V (lateral X ray picture) the spike of Valls's instrument, but also the wire which has been introduced by this instrument with Fuchsig's modification. The spike might prevent rotation of the head during the driving in of the nail.

The only objection to the guide wires is that they may bend and then cannot be withdrawn. The reviewer has had an instrument made on the design of Felsenreich which has never failed to withdraw a wire if unavoidably bent (Figure VIII); he also has used the Valls guiding instrument and broad Felsenreich nail.

THOMAS KING.

THE USE OF MORPHINE AFTER ABDOMINAL OPERATIONS.

Olof Sjöqvist: "*Über die Verwendung von Morphin nach Bauchoperationen: Eine pathologisch-physiologische und klinische Studie*", *Acta Scandinavica Chirurgica*, Volume lxxviii, May 27, 1936.

SJÖQVIST discusses the question whether morphine should be used in the after-treatment of abdominal operations or should be prohibited. The answer to this question depends partly on the opinions of how morphine acts on the human intestine, and partly on the conception of so-called post-operative intestinal paresis.

Since 1928, in the surgical department of the Serafimerlazarette, in all abdominal cases, and especially those in which so-called intestinal paresis was threatened, expectant treatment has been adopted; that is, the patient is treated with rest, with rather large doses of morphine; and no bowel washouts, enemata or drastic purgatives are given.

Intestinal movements are of two kinds—peristaltic movements and pendulum movements, the so-called rhythmic contractions. The peristaltic movements are for the transport of material in the bowel. Stimulation for these movements originates in the intestinal lumen and the reflex mechanism lies in the intestinal wall itself. True peristaltic movements do not occur in the empty intestine; their function is therefore plain—the transport of food material. Pendulum movements are rhythmic movements which occur in the intestine, and which persist even after it has been completely isolated from nervous connexions. Their frequency is increased by heat and by stimulation of the vagus, and decreased by stimulation of the sympathetic. They are regarded by some as arising myogenically, and by others as originating neurogenically in Auerbach's plexus. They occur in the empty intestine.

Usadel suggests that these pendulum movements have something to do with promoting the circulation of the intestinal wall. This author points out that there is no sucking action where the liver veins open into the *vena cava*, because experiment shows that in this spot the pressure is continuous and positive, and that, therefore, the blood cannot be aspirated to the liver by respiratory movement. He showed that the flow of portal blood in the intestinal wall was a function of these pendulum movements. He introduced a current meter into the portal vein of a dog, and by rinsing the intestine with hot water and causing lively pendulum movements, he was able to obtain an increased rate of current in the portal vein.

Sjöqvist has, for many years, been making a series of experiments on human intestinal muscle strips obtained from intestinal resections. He finds that morphine increases the tone of the longitudinal musculature of the small intestine, and also that of the large intestine. The frequency of the tonus swings, and to a lesser extent the pendulum movements are somewhat increased. Morphine acts on the circular musculature by causing a frequency increase of the rhythmic contraction, while the tonus is influenced to a lesser degree. Large doses of morphine —much larger than those used therapeutically—caused the preparation to become fixed in a massive contracted position; no paralysing effect from morphine could ever be demonstrated in these preparations from the human intestine. "Opio-

total", a preparation which contains all the principles of opium, behaved differently from morphine when applied to these preparations of human intestine. After a short inconstant excitation, the preparation was paralysed by "Opio-total"; that is, its tonus sank and the rhythmic contractions were lessened or ceased. Sjöqvist believes that this action of "Opio-total" is due to papaverin, for the drug papaverin itself, even in a much smaller dose than that contained in the "Opio-total", when applied to the intestinal preparations, exercised the same action.

Sjöqvist states that it is high time that the old erroneous belief that morphine acts primarily by paralysing the intestine should be allowed to lapse. The constipating action of opiates is explained (*a*) by the different action of morphine from that of opium, (*b*) by its action in causing delayed emptying of the stomach, (*c*) by the reduction of the defæcation reflex, (*d*) by causing a lessened peristaltic excitability.

Sjöqvist thinks that the expression "post-operative intestinal paresis" is a misnomer, that intestinal paresis is an assumption and that its existence has never been proved. He thinks that the term "post-operative distension" is more appropriate.

Sjöqvist believes that the post-operative intestinal condition cannot be caused by a vasomotor paralysis, because Olivecrona showed that in peritonitis after death of an animal the vasomotor centre was capable of reaction.

Sjöqvist argues as follows: A certain amount of post-operative distension follows every laparotomy, even when the abdominal interference is slight and the patient experiences no discomfort. This distension could not be the result of an intestinal stasis in consequence of the weakness of the transporting strength of the intestinal muscle, because in the same patient when not operated on, an intestinal stasis occurs—no passage of gas or fæcal material takes place for days—and there is no abdominal distension. Further, he reasons, post-operative distension soon disappears. Where does the gas go? Comparatively little of it passes by the anus, so most of it must be resorbed and discharged by the lungs.

Sjöjvist thinks that the increased volume of the intestinal contents is the result of transudation from the intestinal wall of carbon dioxide, which is caused through the weakening of the circulation in the splanchnic area. Gas exchange in the intestine, he thinks, follows the same laws which govern gas exchange in other tissues of the body. If the capillary blood in the intestine is over-saturated with carbon dioxide, some of it diffuses into the lumen. If the carbon dioxide content of the capillary blood is again reduced as a consequence of improved circulation, excess of carbon dioxide in the lumen of the bowel becomes resorbed. His investigations show that it is possible for carbon dioxide gas in the intestine to be resorbed into the blood and excreted by the lungs, as well as being passed by the anus.

Sjöqvist thinks that the aims in the treatment of threatening or of existing post-operative distension are clear. They must be: (i) The greatest possible reduction of those reflexes which bring about the intestinal immotility and which reduce the tone of the blood vessels; (ii) the keeping up of the strength of the circulation by retaining the intestinal muscle movements, especially the rhythmic contractions; (iii) the maintenance of the intestinal tonus; (iv) the strenuous avoidance of unnecessary operative or other trauma of the abdomen, which would increase all inhibiting reflexes on intestines or blood vessels.

In regard to the use of morphine, Sjöqvist quotes Plant and Miller's observations on thin-walled herniæ, in which the movement of the intestinal walls could be easily seen through the thin walls of the hernia. These experiments show that, after the administration of doses of morphine, there is a lively motility of the human intestine in so-called post-operative intestinal paresis.

Most Swedish surgeons, he states, have given up high washouts and high enemata, on the principle that, as most of the gas passes not *per anus* but through the blood, they are valueless. Neither the patient nor the nurses in his opinion have missed them.

For the first twenty-four hours after operation, when there is an insufficiency of abdominal pressure, brought about by the pain of the wound, he uses a rectal tube to counteract the effect of the uninjured *sphincter ani*. He finds that purges and saline aperients make the patient worse.

Sjöqvist will not use remedies such as acetylcholin, physostigmine, pilocarpine, pituitrin extracts, peristaltin or prostigmine. He thinks the action of the first three is that of a poison which acts on the whole para-sympathetic system, and, if given in amounts sufficient to produce an action on the intestine, they give rise to unpleasant by-effects. Given in amounts small enough to avoid these unpleasant effects, they have very little action on the intestine. He does not use them at all and thinks that the value of their action is open to doubt.

Sjöqvist's present methods of treatment, in vogue since 1929, are as follows:

1. No aperient is given before the operation, except in cases in which an intestinal resection is planned. An aperient may be given a good while before the operation, but so long before that the intestine will be in a state of rest at the time of the operation.

2. An enema is given on the evening before the operation.

3. A dose of 1·0 to 1·5 centigrammes of morphine is given the evening before the operation.

4. The anæsthetic used for a short operation is ether inhaled from an open mask; for a long operation gas and oxygen are used.

After operation his routine is as follows:

1. Administration of morphine is given as follows: (*a*) Morphine is given when the patient wakes up after the operation. (*b*) A dose of 1·0 to 1·5 centigrammes is given the evening after the operation. (*c*) This dose is repeated in the morning if the patient is not sleeping, or if he complains of pain.

2. During the day following the operation, warmth is applied to the abdomen by warm water bottles, or some form of electric hot compress.

3. A small hard gum-elastic catheter is introduced into the rectum, and left there during the first day.

4. Morphine is given each evening for the first three days after the operation, and at other times if the patient complains of pain or of abdominal distension. Sometimes 6·0 centigrammes of morphine have been given in the day, and on several days 3·0 to 4·0 centigrammes a day. No disadvantages have been noticed following the administration of this amount of morphine. When the abdominal condition is normal, the morphine is reduced in amount or discontinued. Sometimes it is necessary to give a small dose of "Opio-total" or "Pantopon" during "disaccustoming". Morphine is never given in operations on the stomach, because it produces a contraction of the pyloric sphincter (Magnus, Von den Velden, Tazner and Turold). Instead of morphine "Opio-total" or "Pantopon" is used.

5. On the fourth to the sixth day after the operation, according to the smoothness of the post-operative course, an enema is given. It is often found, as a result of this regime, that a spontaneous bowel action may take place, sometimes even a few days after the operation. As soon as the post-operative vomiting has stopped, the patient is allowed to drink as much as he likes. Fluid, if necessary, is administered by the intravenous subcutaneous method.

H. B. DEVINE.

SERUM THERAPY OF APPENDICITIS.

E. Urech (Lausanne): "*Appendicites et Sérothérapie*", *Schweizerische medizinische Wochenschrift*, Volume lxvi, December 26, 1936, page 1304.

IN Switzerland five hundred people die of appendicitis each year. Urech thinks it should be possible to improve this death rate. Professor Weinberg, in 1930, in a lecture to the Vaudois Society of Medicine, advocated a method of serum injections for the treatment of acute appendicitis. This method comprises an injection of antigangrene serum into the peritoneal cavity, followed by an injection of the same serum in the left side. The method has been extensively followed in the district of Vaudois University. In memory of Professor Weinberg, Urech has followed, in the department of Professor Vuilliet at the surgical clinic of Lausanne, the results thus obtained since 1930. He has also followed up those obtained from 1930 to 1935 in his own department in the Hospital of the Deaconesses of St. Loup. After a statement of the variations of technique in the years preceding 1930, when serum therapy was practised, variations which rendered difficult the exact com-

parisons of results, he gives the results of the first series of cases in the table from which he draws the following conclusions. From 1928 to 1929, 469 patients with acute appendicitis were operated on as emergencies, with 11 deaths, that is 2·3% mortality rate. In 1930, 313 patients were operated on for acute appendicitis, and of these, seven died, a death rate of 2·2%. He points out that an examination of these figures shows that, in the treatment of cases of acute appendicitis in the department of Professor Vuilliet, serum therapy has not effected a reduction in the death rate. The number of emergency admissions for acute appendicitis in the St. Loup Hospital in five years was 480, and, among these, 15 patients were treated by the injection of Weinberg's serum. Of these 15 patients treated with serum therapy, there were 12 deaths and three cures—20%. The 465 cases treated by appendicectomy have given only three deaths (0·66%), the death rate of the total of 480 cases having been 1·25%.

A detailed study of the results shows that in none of the cases of acute appendicitis treated at the Lausanne clinic or at St. Loup has the employment of serum therapy caused striking modifications of the clinical symptoms, nor has it modified unexpected forms of the local condition. Thus results do not justify a conclusion that serum therapy is of any great advantage in the treatment of acute appendicitis. Urech thinks that improvement of results in the surgical treatment of acute appendicitis will come only by earlier diagnosis and therefore earlier intervention.

P. GRISEL.

Analysis from *Journal de Chirurgie*, Volume I, Number 3.

Reviews.

DIATHERMY.

Diathermy, Including Diathermotherapy and Other Forms of Medical and Surgical Electrothermic Treatment. By E. P. CUMBERBATCH, M.A., B.M., D.M.R.E., F.R.C.P., with nine collaborators; Third Edition; 1937. London: William Heinemann (Medical Books) Limited. Demy 8vo, pp. 582, with illustrations. Price: 21s. net.

THE third edition of this book is so enlarged and rewritten that it can almost be regarded as a new work. Additional chapters by different authors make the work complete.

Chapter III, on physical principles, is a necessary addition, and deals with the principles underlying diathermic treatment without going too deeply for the average reader. With the present vogue of short-wave therapy, familiarity with the principles governing displacement currents is essential. We should, however, in a work such as this, like to see more written in regard to the technique required for the practical application of these principles. In short-wave therapy results depend almost as much on the use of an accurate technique as on a knowledge of physical principles: the treatment end of the circuit is of as much value as a knowledge of how the current is produced.

In this edition the paragraphs are larger, and at the end of each one a short summary is given of the experience of the writer in the treatment of the various conditions discussed.

In the fourteenth and fifteenth chapters will be found the theory and practical application of diathermy in regard to its use in the field of gynæcology and chronic arthritis. Much criticism has been levelled at this form of treatment when applied to diseases of women; but the fact remains that in established gynæcological clinics all over the world it is still employed on lines similar to those originally laid down by Robinson.

Chapter XIX gives a short summary of the electrical methods employed for producing artificial fever in the treatment of *dementia paralytica*. Although not

universally used, this method of treatment is entitled to consideration, because it is safe and easily controlled.

It is to be regretted that the collaborator in ophthalmology has not had experience in using short-wave therapy in pathological conditions of the eyes. It is easy to apply and the results are good.

In the chapters on short-wave therapy and inductothermy will be found fresh information on these important and modern forms of treatment. A bibliography on these subjects is given.

Part III, written by the author and his collaborators, deals with the theory and practice of electro-surgical methods, and gives descriptions of technique in regard to their special application.

With the help of his collaborators, the author has shown how wide is the field of application of diathermotherapy. This book will be found a useful guide to practitioners and technicians.

ORTHOPÆDIC SURGERY.

Handbook of Orthopædic Surgery. By A. R. SHANDS and R. B. RANEY; 1937. St. Louis: C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary Limited. Royal 8vo, pp. 593, with 169 illustrations. Price: 30s. net.

"THE subject matter of the text has been divided into twenty-four chapters in accordance with a report of the Committee on Undergraduate Instruction on Orthopædic Surgery of the American Orthopædic Association, which in 1934 called attention to the fact that in several of the leading medical schools approximately twenty-four hour class periods of an hour's duration are profitably employed for undergraduate orthopædic instruction." The book has been written for the medical student and the general practitioner in order to present the fundamental facts and principles of orthopædic surgery. The twenty-four chapters have been submitted for criticism to twenty-four teachers of orthopædic surgery, and some of these have international reputations. Unfortunately, the book does not deal at all extensively with orthopædic principles as promised by the authors, but rather describes in a short form the diagnosis and treatment of most orthopædic conditions. This may be useful to the general practitioner. The student obtains in this book merely a full vocabulary of orthopædic diseases and disorders, knowledge in concentrated form, which on the whole is described in ordinary textbooks or manuals of surgery. The illustrations are all line drawings, and many have much merit. The actual information in the reading matter is up to date on the whole, but throughout the book treatment is described indefinitely. If this book were used by the students the teacher who prescribed it would no doubt amplify the text, especially in regard to the principles underlying diagnosis and treatment. There are over 500 pages, 169 illustrations, and a bibliography, mainly of books, at the end.

A TEXTBOOK OF SURGERY.

Rose and Carless Manual of Surgery for Students and Practitioners. By C. P. G. WAKELEY, D.Sc., F.R.C.S., F.R.S., and J. B. HUNTER, M.C., M.Chir., F.R.C.S.; Fifteenth Edition, in two volumes; 1937. London: Baillière, Tindall & Cox. Royal 8vo, pp. 1630, with 948 figures and 18 coloured plates. Price: 30s. net.

"ROSE AND CARLESS MANUAL OF SURGERY FOR STUDENTS AND PRACTITIONERS", by Cecil P. G. Wakeley and John B. Hunter, has attained its fifteenth edition. That this standard work should have maintained its popularity as a textbook for students for just thirty-nine years is an indication of the sound lines on which it was originally designed. The present edition appears as two volumes, beautifully bound and called a "Coronation Edition". It has been brought up to date, there is an increase of 130 pages in the text, and there are 242 new

illustrations. The chapters on bacteriology, inflammation and immunity have been rewritten by Carnegie Dickson, and this author has succinctly dealt with many new aspects of these subjects. Of the new matter, the chapter on shock is one of the most valuable in the book. Primary and secondary shock is discussed, and the treatment of shock is simply outlined. Recent advances in X ray and radium treatment have been added to the chapter on the use of physical agencies in surgery. The indications for the use of radium and the methods for its application are described. Another completely new chapter is that on the surgery of the sympathetic nervous system, in which the principles governing the application of sympathectomy for the various diseases for which it has been used are laid down. The section dealing with the surgery of the central nervous system has been revised, and much new matter added. The chapter on thoracic surgery has been brought up to date. The book is beautifully printed, excellently illustrated and well produced. It is a student's manual and complete to a degree. There is no teaching in the book which would be dangerous to the student. Highly specialized surgical treatment has been eliminated. Some conditions and their treatment are extensively treated, perhaps too extensively, for a student's manual. The book can be highly recommended as a standard textbook for the student. It is indispensable for the general practitioner, and this edition should find a place in every surgeon's library, especially those whose duty it is to teach students.

OBSTETRIC OPERATIONS.

Operative Obstetrics: A Guide to the Difficulties and Complications of Obstetric Practice. By J. M. KERR, LL.D., M.D., F.C.O.G., with the assistance of D. MCINTYRE, M.D., F.C.O.G., and D. F. ANDERSON, M.D.; Fourth Edition; 1937. London: Baillière, Tindall and Cox. Royal 8vo, pp. 860, with 338 illustrations. Price: 45s. net.

IN this fourth edition of his well-known work on operative obstetrics Munro Kerr has taken the opportunity to revise the volume and to incorporate the latest advances in the subject. It represents the accumulated experience of forty years' practice of a recognized master of British obstetrics, and of one who in addition is not averse to trenchant criticism of methods with which he is not in agreement.

The keynote of his work, as in previous editions, has been a description of knowing when and how to interfere in obstetrical emergencies. The book commences with a very valuable summary of the modern standpoint regarding the methods to be employed in the prevention of maternal mortality and morbidity. Throughout the book runs a sane and conservative commentary on current methods. For example, while admitting that many authorities have obtained excellent results from rupture of the membranes for induction of labour, he still advises caution in accepting this method to the exclusion of all others.

The chapters on occipito-posterior and breech presentations contain a judicial summary of the dangers of too early interference or of ill-conceived methods of delivery. As may be expected, the description of the treatment of pelvic disproportion and the advocacy of a trial of labour for all *primiparæ* with minor degrees of contraction are models of concise reasoning based on an unrivalled experience at Glasgow. The details of various operative procedures are well presented, as is also a survey of the present status of various anæsthetic and analgesic agents.

A valuable feature of the volume is the extensive bibliography attached to each section. One minor criticism which may be made is that the treatment of septic abortion is rather scantily dealt with, and that the work of Hill, of Melbourne, regarding *Bacillus welchii* infection is completely ignored. This is rather surprising in view of the general up-to-dateness of the literature in all other sections.

This work represents the ripe experience of a great Scottish teacher, and senior obstetricians will appreciate his arguments. No better guide can be recommended to the younger generation, both of specialists and those whose practice

includes much obstetric work. As Munro Kerr states, "Knowledge comes, but wisdom lingers", and on this note we close a review of the most stimulating practical work on obstetrics which has been published within recent years.

OTO-RHINO-LARYNGOLOGY.

Diseases of the Nose, Throat and Ear: A Handbook for Students and Practitioners. By I. S. HALL, M.B., Ch.B., F.R.C.P.E., F.R.C.S.E.; 1937. Edinburgh: E. and S. Livingstone. Crown 8vo, pp. 438, with 55 illustrations. Price: 10s. 6d. net.

THE subtitle and the preface of this book clearly define its limitations, and we must say that we think the author has achieved his objective. The treatment of the subject is clear, concise and unequivocal, and includes the whole range of the specialty of oto-rhino-laryngology. The author is obviously an experienced teacher, and the book contains all the information which a student is likely to require. The information is also very clearly and attractively set before him. In other words, the book may be regarded as a characteristic product of the Edinburgh Medical School.

The illustrations as a rule are very good, with the exception of some of the X ray reproductions, which might have been better as line drawings of tracings. A mild protest may be made concerning the photograph on page 189 illustrating the method of carrying out indirect laryngoscopy. Surely a better method of holding the tongue is between the thumb and third finger with the flexed index finger controlling the upper lip.

The production of the book from the publishers' point of view leaves nothing to be desired.

OPERATIONS ON THE EYE.

A Text-Book of Ophthalmic Operations. By H. GRIMSDALE, M.B., F.R.C.S., and E. BREWERTON, F.R.C.S.; Third Edition; 1937. London: Baillière, Tindall and Cox. Medium 8vo, pp. 331, with illustrations. Price: 17s. 6d. net.

IN the preface to this the third edition of their well-known textbook on ophthalmic surgery the authors state that they "have excised much that had interest only in the development of operative surgery, and have tried, by leaving out unessential matter, to make a book more helpful to the student of ophthalmology". Since in the practice of a large number of oculists operative surgery plays a minor rôle and the opportunities for gaining surgical experience are limited, the aim of the authors is a laudable one. It is doubtful whether they have gone far enough in this direction, as there is much in some sections, particularly those concerned with the surgery of the muscles and eyelids, to confuse the student.

In discussing the treatment of strabismus the authors, perhaps wisely, make no reference to orthoptic training. The description of the various operations is lucid and well illustrated. Nevertheless, when the reader arrives at the end of the chapter, he is still undecided as to which procedure will suit his needs. In our experience most cases of concomitant strabismus can be adequately treated by the judicious use of the operations of resection and recession. It is surely time that there was some standardization of operative procedure in these cases. In view of the present popularity of tenectomy of the inferior oblique, it is strange that there is no description of the operation.

Measures designed to alleviate the entropion and trichiasis due to trachoma are described at length. Many of these are of historical interest only, and the technique of Van Millingen, if carefully followed out, will be followed by prompt and permanent relief of the condition.

Wisely enough, much more space has been given to the description of extracapsular extraction of cataract than to the intracapsular operation. Prefer-

ence is expressed for the method with peripheral iridectomy. There is no mention of the routine use of retrobulbar injection, and the sane outlook of the authors towards an operation so greatly feared by the inexperienced should do much to reassure the surgeon who prefers the preliminary iridectomy. Confidence is half the battle in intraocular surgery and the over-cautious attitude as evidenced by the routine use of retrobulbar injections is to be deprecated. The value of a preliminary iridectomy as a safety measure is doubtful.

Iridectomy for acute glaucoma is carefully and fully described. Whilst the authors appear to prefer a narrow Graefe knife to the keratome, the advantages of the former are not sufficiently emphasized. When opportunities for experience are few, there is no doubt which is the safer instrument. The remainder of the section on glaucoma is carefully compiled.

The more recent methods for treating detachment of the retina and opaque corneæ are rather summarily dealt with. The authors, no doubt, feel that operative measures designed to treat these conditions are still in a state of flux and that insufficient time has elapsed to allow for a competent judgement of their respective merits.

Comprehensive reference books on ophthalmic surgery are rare in the English language, and this one may be confidently recommended to the ophthalmic surgeon.

Notices.

THE BRITISH POST-GRADUATE MEDICAL SCHOOL.

THE College wishes to draw attention to the announcement of the British Post-Graduate Medical School on page xxx of the advertisements.

NEW DEVELOPMENTS IN SURGICAL EQUIPMENT.

THE attention of Fellows is drawn to pages xviii and xxvi among the advertisements, which illustrate some recent developments in surgical equipment. The Editorial Committee is responsible for the selection of the equipment illustrated thereon. The publishers will be pleased, whenever possible, to supply the names and addresses of the manufacturers to anyone requiring such information.

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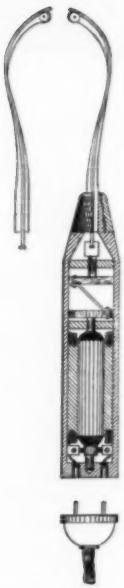

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(*Journal of Bone and Joint Surgery*, Volume XIX, Number 1, January, 1937, page 246.)

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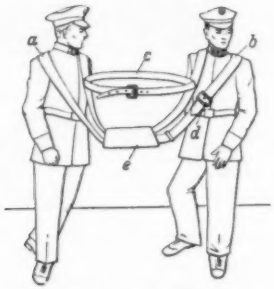

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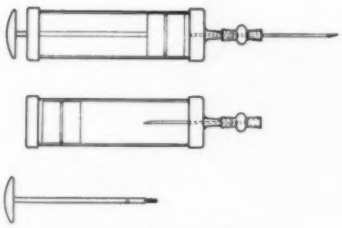
(*Der Chirurg*, Volume IX, No. 8, April 15, 1937, page 320.)

The chair consists of a firm unpierced canvas seat hung from two sling-shaped shoulder straps (*a*) and (*b*). The rear ends of the shoulder straps are connected by a girdle (*c*). The width of the girdle is adjustable by a buckle. Both shoulder straps are adjustable in length by the buckles (*d*). It is therefore not necessary to have attendants of the same height and both attendants have their two hands free.



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(*Der Chirurg*, Volume IX, Number 8, April 15, 1937, page 319-320.)

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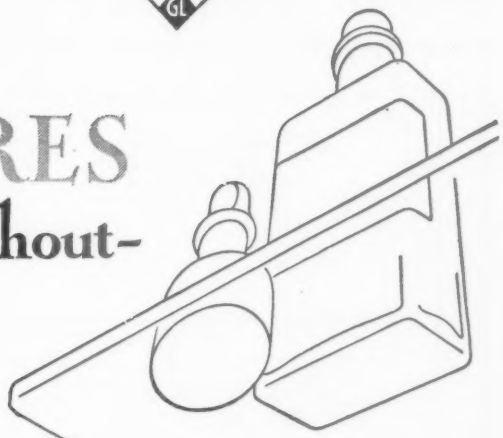

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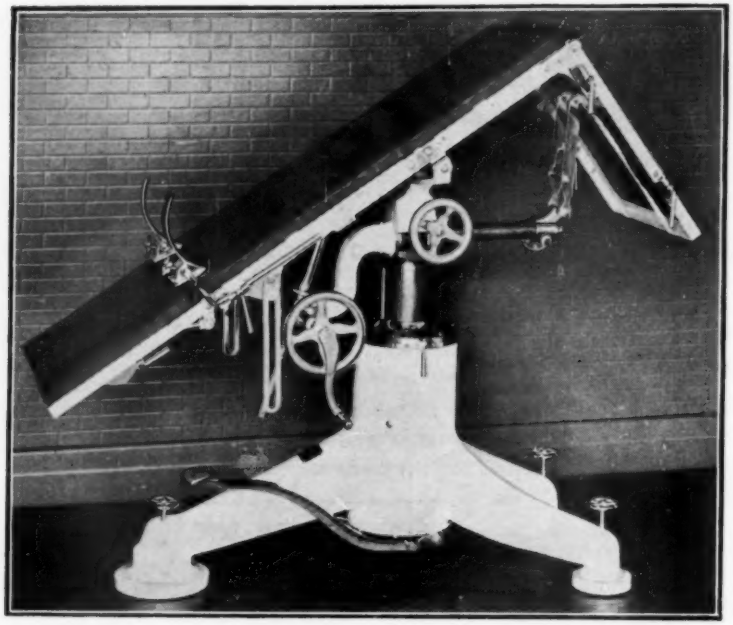

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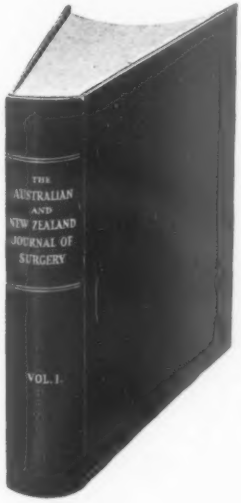

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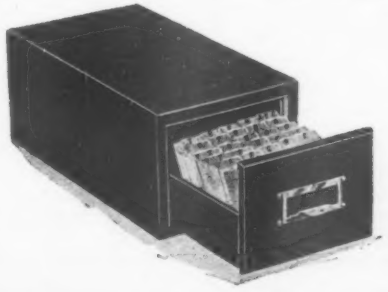

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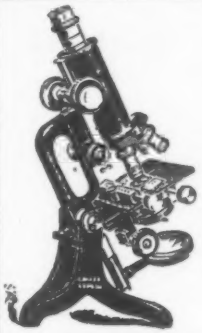

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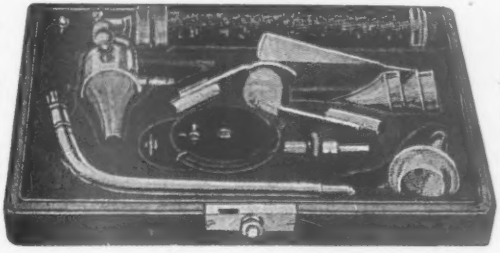

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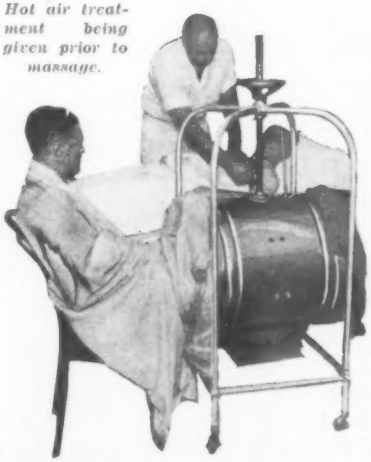

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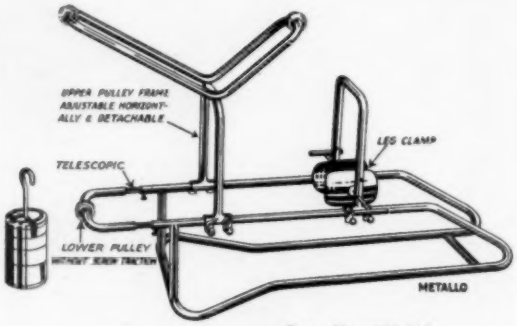

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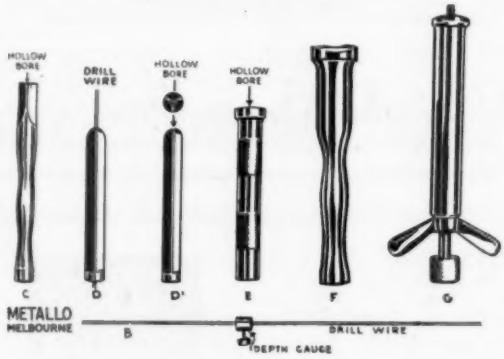

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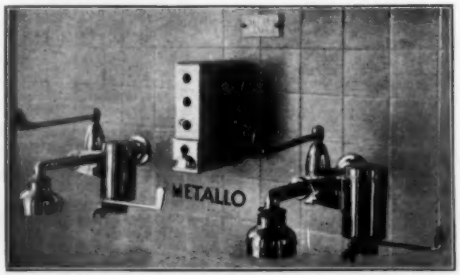

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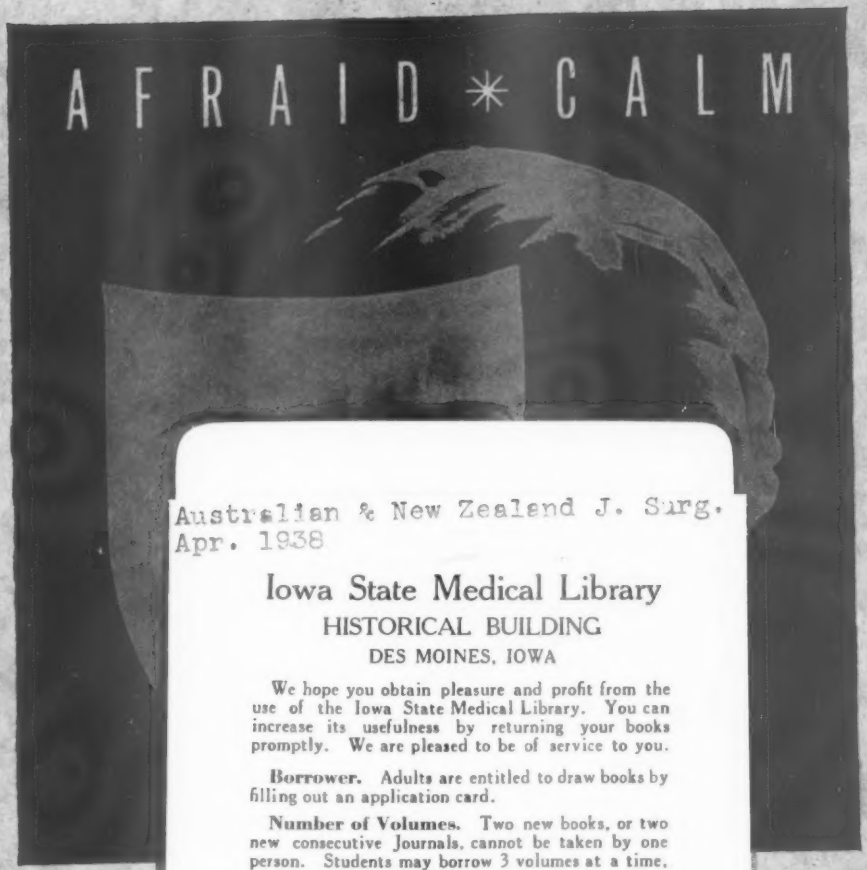

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